ROYAL COMMISSION ON MATTERS OF HEALTH AND SAFETY ARISING FROM THE USE OF ASBESTOS IN ONTARIO

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Dr. Brian Gibson, Toronto Occupational Health Resource Committee
Mr. Ray Stone, Government of Ontario

180 Dundas Street
Toronto, Ontario
Wednesday,
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VOLUME XII

ROYAL COMMISSION ON MATTERS OF HEALTH AND SAFETY

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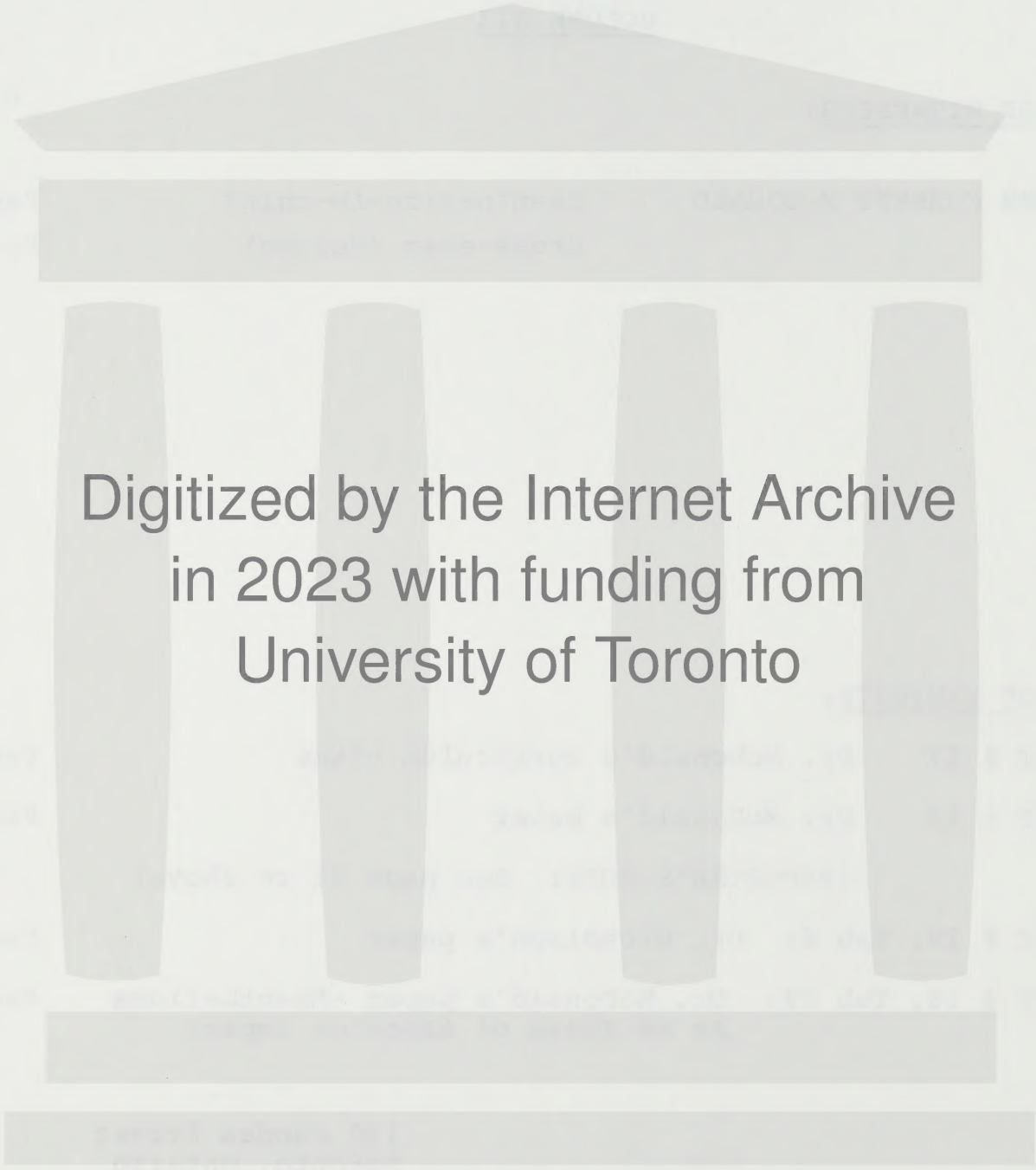

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THE FURTHER PROCEEDINGS IN THIS INQUIRY RESUMED PURSUANT TO ADJOURNMENT

APPEARANCES AS HERETOFORE NOTED

DR. DUPRE: Before I greet our guest, are there any matters you wish to raise?

MR. LASKIN: There are some new faces with us this morning, Mr. Chairman. Perhaps I could introduce those people who are with us for the first time.

On my left is Mr. Ray Stone, who is assisting Mr. McNamee and will be here for the Government of Ontario today.

Behind me, Mr. Philip Casgrain.

MR. CASGRAIN: If I may take this opportunity, Mr. Chairman, to wish you on this very auspicious day in Quebec, with my wife and everybody else who comes from there, a happy Jean Baptiste Day to you all.

MR. LASKIN: Mr. Mark Nelson is with Linda Jolley and is acting for the Ontario Federation of Labour and is with us today.

Mr. Brian Gibson, behind us, has been at some of our meetings and is here today from the Toronto Occupational Health Resource Committee, Mr. Chairman.

DR. DUPRE: Any other matters that you or your any of your colleagues have, counsel?

MR. LASKIN: I don't ...

MR. GIBSON: I don't have any other matters.

MR. LASKIN: I don't believe so, thanks, Mr. Chairman.

DR. DUPRE: Well, may I please, on behalf of all of us, greet most warmly our distinguished visitor today, Dr. Corbett McDonald, who has kindly come a long way to give sworn testimony before us.

Dr. McDonald, you are most gratefully welcomed here, indeed, sir.

DR. McDONALD: Thank you, Mr. Chairman.

DR. DUPRE: Miss Kahn, would you swear in the witness, please?

DR. JOHN CORBETT McDONALD, AFFIRMED

EXAMINATION-IN-CHIEF BY MR. LASKIN

MR. LASKIN: Mr. Chairman and Commissioners, I have distributed to everyone, including yourselves, a copy of Dr. McDonald's curriculum vitae, which I propose to file in evidence. But you will note, and Dr. McDonald I take it you were at one time Chairman of the Department of Epidemiology and Health and McGill University, and then went to London, England, and I take it you have been with the London School of Hygiene and Tropical Medicine during your stay in England?

THE WITNESS: A. Yes, for five years.

MR. LASKIN: Q. I understand, happily for Canada, you are returning to this country in the fall?

THE WITNESS: A. Yes.

Q. Are you coming back to McGill University?

A. Yes.

MR. LASKIN: Might I file as the next exhibit...

MISS KAHN: Exhibit sixteen.

MR. LASKIN: Sixteen. Might I file as exhibit number sixteen Dr. McDonald's curriculum vitae, which lists his

MR. LASKIN: (cont'd.) very numerous publications, a great many of which are in the field of the health effects of asbestos.

EXHIBIT # 16: The abovementioned document was then produced and marked.

MR. LASKIN: Might I also file, Mr. Chairman, as exhibit seventeen, a copy of the brief containing a selection of Dr. McDonald's publications, numbering twenty-seven.

EXHIBIT # 17: The abovementioned document was then produced and marked.

THE WITNESS: I would like to comment on that.

MR. LASKIN: Yes, by all means.

THE WITNESS: Can I say that some of these are not my publications. Included in here, I think, are two or three which are...

MR. LASKIN: Are commentaries on some of your publications, correct.

THE WITNESS: Yes, that I was not aware of.

MR. LASKIN: Fair enough.

MR. LASKIN: Q. Dr. McDonald, as I read through the publications which you authored or coauthored, it seemed to me that the two broad areas of your research, or at least two main areas of your research were, first of all, a continuing and indeed comprehensive study of the cohort of some eleven thousand-odd persons who were all of those employed in the Quebec chrysotile production industry, and born between 1891 and 1920, and employed one month or more. I took it that was one broad area in which you have done a considerable amount of work with your colleagues.

The second area, I gathered from your research, was a North America-wide, indeed a worldwide investigation of the

Q. (cont'd.) incidence of mesothelioma. Would that be putting it fairly?

A. I think it would create the impression that there were two lines of research, whereas I don't think that's quite true. I think our studies in the Quebec mining and milling industry included a cohort study and this was only one aspect. I think it does need to be seen in context.

The other point is that equally, our studies of mesothelioma have by no means been confined to case ascertainment in North America.

Q. I want to develop some of the issues that arise out of your work, and I wonder if I could start with just a general question.

As I understand it, you have used two main methods of analysis - a so-called man-years approach which you call an a priori approach, and I take it that means going from cause to effect, and a case-controls approach or a posteriori approach, as I think you call it, going from effect to cause, I take it? Am I phrasing it accurately so far?

A. Yes.

Q. I gather from your writings that you have given a considerable amount of thought to the strengths and weaknesses and the advantages and disadvantages of these two kinds of approaches, especially when you're dealing with a subject such as the health effects of asbestos, and I wonder if you might just at the beginning be good enough to sketch for us in perhaps a general way what you see as the advantages and disadvantages of these two kinds of approaches in analyzing this problem?

A. In epidemiological studies of cause and effect, we are in essence looking for association between cause and effect. You could say there is an association, you can either say a certain people...a certain proportion of subjects who have been associated with some form of exposure to some environmental

A. (cont'd.) factor will develop disease. You can equally take persons who have a disease and persons who have not a disease and compare the proportions of those two groups that have had an exposure.

In certain circumstances there is absolutely no difference between those approaches. That is to say, you are using precisely the same data, but you are serving it up in a different way.

Now, forgive me if I'm a bit lengthy on this because it's a very big subject.

If you know everything...let's say if you take a defined group of persons and follow them in time, and identify what happens to them all, you can analyze that data either way, in the way I've just mentioned. You can either say what proportion of persons exposed, shall we say to asbestos, developed trouble, compared with those that were moderately exposed or lightly exposed or not exposed in that group, or you could turn around at the end and take within that defined cohort of persons all the diseases that occurred and compare, let us say, because this is relevant, the cases less the deaths from asbestosis, deaths from lung cancer, and look at their experience of exposure compared with others who did not die of lung cancer or asbestosis.

Now I want to emphasize, therefore, that the two approaches, if you know everything, in fact are the same. You can work one way and get the other answer by simply turning it around. It's purely an arithmetic game.

However, the a posteriori method, which is essentially taking the cases and comparing the experience of cases with noncases, is more flexible. In analysis you can compare the characteristics of the cases in respect of a larger number of factors than you can the other way around.

A. (cont'd.) You can do it the other way on, but in fact it is really quite difficult and it is largely for that reason that one introduces it.

However, there is an entirely different aspect to all this, and that is in which first of all the orthodox way of analyzing a cohort study...I take it that everybody is clear what I mean by that...is to do the man-years approach and to essentially compare persons with different levels of exposure for different intensities, different durations, to compare their rate of occurrence of disease, and at the end of the day to use, or rather in that process, to use a reference population, so-called very commonly, the experience, the mortality experience of the general population of the area in which you are, the province or the country.

In many studies, epidemiological studies, you don't really know anything about the exposure. In fact, one of the characteristics of most asbestos studies is that there has been no effort to determine the amount of exposure. And in those situations what you are really doing is to compare the mortality in the exposed occupation with what would have been expected if the rates of mortality in the general population had applied.

Now when you do that, there are real problems. It's the orthodox thing to do, but if you think about it for a moment there is no particular reason to think that a specific occupational group will have the mortality experience of the general population. They are selected by wanting to do the job, they are selected by the employers who employ them, they are selected by the part of the country, they are selected in relation to economic and social factors, a hundred sort of selective factors which mean that they are different from the general population.

It still allows you to use this general population experience, providing you don't think they should be the same. It is still a very useful basis for working out

A. (cont'd.) your rates.

Now, one of the big advantages of the case-control or a posteriori approach, is that it doesn't make any assumptions of that kind. It doesn't need...you don't go outside the population that you are studying. You don't have to make any...you make no use of the general population at all. You simply compare the distribution of exposure, or whatever it is, in cases of the disease, with those who didn't. What's the difference between the person who gets lung cancer in an industry and a person who doesn't? You examine smoking, duration of employment, intensity of employment, type of work, anything you like.

Now, there is one third problem. I think that most statisticians, epidemiological statisticians...I mean to say this whole thing has had a lot of airing and discussion recently in scientific circles...I think what I said would be generally agreed. I don't think there is any important controversy about the fact that within a cohort it doesn't make any difference which way you do it, but it may sometime be both convenient and more sensitive and more flexible to use the a posteriori method.

But we have to bear in mind that case-control studies and analyses are also done in which you do not have a cohort. That is to say in which you get a series of cases of something, and say I've got a series of cases of people who are blind in one eye and I want to know whether it might be due to their work. So then we have to sort of take a series of cases of people blind in one eye and a series of comparable people who are not blind in one eye, and see if they had a different type of work history.

Now, in that situation you do have real difficulty in deciding what would be a fair comparison, and I think some of the misunderstanding about case-control analyses arises from the fact that case-control designs of study for epidemiology are sometimes open to criticism on the grounds of

A. (cont'd.) bad selection of controls.

I hope that's told you something.

MR. LASKIN: Q. Good. Thank you very much.

Can we move now to your own work and can we start with your most recent, or one of your most recent papers on your Quebec cohort, and it's the paper at tab eighteen of exhibit seventeen, Dust Exposure and Mortality in Chrysotile Mining, 1910 to 1975, the paper that appeared in the British Journal of Industrial Medicine in 1980.

THE WITNESS: A. All right.

Q. Do I take it this particular cohort, the Quebec miners and millers, you have now studied to four points in time - 1966, 1969, 1973 and now, finally, 1975?

A. Yes.

Q. By 1975, I take it, nearly half of your cohort was no longer alive?

A. Right.

Q. Might I, Dr. McDonald, first of all just ask you some general questions about methodology, and can I turn first of all to page thirteen of the paper? I just wanted to ask you one or two questions about dust measurements.

I note in the first full paragraph on page thirteen, your indication that you had identified some five thousand, seven hundred and eighty-three jobs and it would appear against that had done dust exposure estimates based on about four thousand midget impinger dust counts. Can I ask you, does that mean that there were some jobs for which there was not an actual measurement made?

A. You could say that all the jobs had no measurement made, because the dust measurements were not made of jobs. These were dust measurements, essentially area samples made in the industry, mainly in the mills rather than the mines, and over a period of time, and Gibbs, Dr. Gibbs, who was

A. (cont'd.) responsible for this work, spent a lot of time in plotting out the distribution of these counts, their location, the year, and as it were, without reference to the jobs, built up his best available picture of what the dust levels were in different parts of the...particularly the mills of Quebec...over the years in question.

I don't recall when the first count was, but it probably was of the order of 19...it wasn't very much before 1950.

So then from that he was able to make an estimate of the pattern of levels. He also interpreted these in the light of all information available to him, such as the information given by workers who were there at the time. For areas that perhaps there was no measurement, he might have well asked somebody to compare one particular part of the mill with another at that time. So it is not...if you like, all these four thousand measurements were pegs on which the hat was hung, and building up...then having done that, we then took the jobs, he took the jobs, and said right, this man was working at such-and-such a job on such-and-such a floor of such-and-such a mill from year to year, and thereby allocated his estimate of what the prevailing dust concentration was. That was the method.

Q. In other words, he took the job and placed it in a particular area...

A. That's right.

Q. ...of a plant?

A. That's right.

Q. How many plants were there, do you recall?

A. Well, if I recall correctly, some of the older papers will give you more on this...I think there might have been in the twenties, maybe twenty to thirty owners originally, who would have, at some point, have come into our study. But by the time we really were properly into the study, I

A. (cont'd.) think they would be of the order of six companies. A seventh, I think, more or less went out of business about the time we started in 1966.

Nevertheless, of course, we included everybody, regardless of what company they had been in. The pattern generally was that smaller companies either went right out or were bought up by larger...there was amalgamation of companies. For the most part, they took over the work history records, the employment records, of the constituent groups.

Q. The second question I wanted to ask you about dust measurements was this: When Mr. Berry gave evidence last week, he talked about the dust measurements that were made at the Rochdale plant that he had studied, and one of the papers that was put to him was a recent paper by Peto, in which he indicated a change in the method of measurement from static sampling to personal sampling, and that was one aspect of it. The second aspect was going to graticule counting, as I understood it, changed significantly the kinds of measurements that were prevalent at Rochdale, and I'm just wondering whether...first of all, what kind of measurement was made in Quebec, and secondly, would there be any kind of difference based on that kind of approach?

A. In Rochdale, because they were not only looking for...they were aiming at a dust exposure level based upon fiber concentrations, and in order to get to that point, they used, again, all available data. As everywhere else, only in recent years have there been any form of direct fiber count measurements. If I remember correctly, there have been difficulties over the standard form in Rochdale, of fiber counting.

Equally, the dust particle counting in Rochdale was different from the one in Quebec. I'm just putting that on one side, because they are not directly comparable.

A. (cont'd.) In Quebec, however, the dust counts which we used in our basic studies were dust particle counts made from about 1950 onwards by the same engineer, Lachance, throughout. Mr. Lachance did virtually it all with the midget impinger, and the standard method of, with all its faults, and there are many, of particle counting was followed throughout.

Now, in more recent years we have been working hard, and particularly my colleague, Graham Gibbs, has been working very hard at attempting, for the Quebec exposures, to move over to what these particle counts meant in terms of fiber concentrations, and again, the man you should ask about the details of that would be Dr. Gibbs, but he has had...he has done it within his own standard methods and has used the counts of other people, fiber counts again, according to the same methods, so that I think in our efforts to convert from particle counts to fiber counts...we may have lots of troubles, but I don't think we've got serious troubles of varying methodologies.

Q. I'll come back to...

A. We have lots of problems, but not that one.

Q. Fair enough.

Can I ask you just one final question about dust measurements? I wasn't clear from your paper, between 1966 and 1975, when you followed through on the cohort, were you continuing to take measurements over that period?

A. It so happens we were, but that is not...we didn't use them in this paper.

Q. I see.

A. For another purpose, yes.

Q. But were employees continuing to contribute man years at risk from 1966 to 1975?

A. Yes, oh yes. A person contributes man years at risk from the moment that he joins until he dies, wherever he has worked. He is at risk because he is at risk of dying. It's

A. (cont'.d) irrespective of whether any measurements have been...

Q. Were the employees also continuing to accumulate dust?

A. If they were still employed, yes.

Q. In terms of your own calculations, of your own measurements?

A. Not for the purpose of our analysis.

Q. Not for the purpose of your analysis?

A. Because we excluded all exposures within nine years, the first nine years. We used a latent period of nine years as the amount of time that we would not include as contributing to their exposure.

Q. That was throughout?

A. It was partly...not exactly forced on us, it was there because we had no work histories after 1966, and didn't feel they would be worth using anyway, because we were satisfied that it was very unlikely that any dust accumulated in the last few years of life would make any difference.

Q. I see. I was just going to...my next question was to ask you what the thinking was behind the nine year limit.

A. I think most people will make some sort of arbitrary decision on this. As a matter of fact, it really wouldn't matter for practical purposes, because even if we had added it in, the pattern of dust concentrations in this and most other industries is such that it fell away really fairly acutely. The levels between 1950...I think the average particle count in Quebec mills in 1950 was around about seventy million particles per cubic foot. I don't know what the current figure is, but I would be very surprised if the current level was averages above one or two, something of that order I would think.

So you can see it has come right down and

A. (cont'd.) therefore anything which, the amount of exposure which would have resulted since 1966 would be absolutely trivial compared with what had gone before, even if we thought it had any biological effect, which I think most people would say it couldn't have.

Q. One final methodological question I just wished to ask you is, I note that you haven't placed any confidence intervals or probability statistics on any of your data, unlike some of the other studies which are around, and I just wanted to ask you what the thinking was behind that approach?

A. This is a philosophical issue, I suppose. But we would think it was quite inappropriate to use tests of significance on observational data of this kind. You also have got to bear in mind that what we are looking at is the entire experience of an entire industry, virtually. We have no sampling, we are not extrapolating..

Q. You've got it all?

A. We've got it all. There it is. There's the data. You can then interpret it as you feel. We would feel, I think, and I would feel quite strongly, that tests of significance on this kind of thing are quite wrong.

DR. UFFEN: Counsel, could I make sure I understand this?

A few minutes ago we saw that the exposures were estimates based on a single individual's lifetime. Wouldn't it be useful to try and get some measure of the range of reliability of those data?

THE WITNESS: Well, I don't know whether it would. I don't know what I would do with it if I had it, shall I say.

The...I may say that we have got a young man working on this for the last three or four years, attempting...what we are concerned with in particular is what is the effect of

THE WITNESS: (cont'd.) random error in measurements.

Now I may say that, of course, what we are much more worried about is not random error, but biased error. But random error, he has been studying and looking at the effect that such random error in dust assessments would have in the shape of the dose-response relationship, and using as his measure of error the rather scanty data that there are on duplicate measurements. That is, say, where the same situation is measured more than once.

So, if you like, from a methodological point of view we have looked to get some sort of idea of what that kind of random error might do to our exposure response. We reckon that it might...I think a large body of statistical theory exists already on this point, namely that any error which is random will in fact decrease the slope of the exposure-response curve.

We reckon, he reckons, that it would be of the order of about sixteen percent, on the data we have. But I do repeat that even this...this is for an academic degree, and what we are much more worried about, really, is not the error so much as the absence of information, or even biased information. I don't know how you would correct for that.

DR. UFFEN: I'll perhaps come back to this some other time, because what is on the back of my mind is, if you go to another region, another area, or somebody else is making the measurements, would this explain apparent differences that we read about in different parts of the country?

THE WITNESS: Well, I mean, I could answer that. Of course it will make a difference if you don't measure the same thing in the same way...you'll get a different answer.

On the other hand, there is no way of knowing how much difference it will make unless you have both those readers measuring the same conditions somewhere so that you have a measure of it. But obviously it will, indeed, and I'm sure

THE WITNESS: (cont'd.) that even although we had one person, Lachance, doing a measurement over twenty years, or whatever it was, he would not be absolutely standard from day to day or from year to year. I presume that he learned something over the twenty years work, and I would expect there to be changes.

But if you are also referring, if you like, to the difference in risk observed in mines as opposed to other situations, my own, no doubt will come to that, but my guess is that it is not a measurement problem for the most part, it's something else.

MR. LASKIN: Q. Just one question about terms before we move to a specific finding.

In talking about causes of death, you used the term pneumoconiosis. Is that equivalent to asbestosis for the purposes of your study, or is it something slightly different?

THE WITNESS: A. I think that, again, I'm speaking from memory now, I think that virtually every death ascribed to pneumoconiosis and coded to pneumoconiosis, something like all but two or three where the word asbestosis appeared.

Certainly we intend it to mean that, but we have included, I think, two or three deaths where it stated pneumoconiosis and not asbestosis, but we have counted it as asbestosis.

Q. And one final question about terms. I know that you, in your cohort, found very few cases of deaths by mesothelioma, but to the extent that they did occur, how are they listed under causes of death...and for example, if you go to page fifteen, table two, what category would they be under?

A. They may get a little bit...I was asked this question just the other day. If I can find the page, we do say somewhere where they are listed. Do you mind if I just look and see if I can find it?

Q. I'm sorry. I don't think I'm in...okay.

A. I seem to recall that six of the ten were listed as lung cancers...

Q. I'm sorry. It's in your text, Dr. McDonald. I just noticed it at the bottom of page...

A. The other four, I think, were categorized as benign tumors, and we, on looking at them, decided that they were mesotheliomas.

Q. So it would appear under All Other Known Causes?

A. It would, I think, have appeared under... yes, it would, I think, because we didn't have a benign category. It would have been under Other Known Causes, yes.

Q. Can I turn now to some of your major findings, conclusions, and right on page eleven, in the abstract, you make a calculation of...we're still at tab eighteen...of overall excess mortality, two percent at Asbestos, and ten percent at Thetford Mines?

A. Yes.

Q. Do I take it that is what it appears to be, an overall figure without regard to specific causes of death and without regard to latency or cumulative exposure or age?

A. This is certainly the crude figure. It is the, what would be called the standardized mortality ratio. It is the observed deaths over the expected deaths, based upon Quebec mortality rates.

Q. Would you expect that figure to rise at all from 1975 onwards, in light of the fact that nearly half of your cohort was dead?

A. No. The more relevant data on this...in fact this paper should be taken in parallel with a paper we gave the year earlier in New York, and it's also listed. Can I refer to it?

Q. Yes, by all means. It's tab seventeen.

A. Tab seventeen. Because some of, if you like, the more crude picture, the general picture, is presented in that paper and I would like to draw attention to, in that paper, this is Mortality in Canadian Miners and Millers Exposed to Chrysotile, there is on page three of that paper a figure one, Observed and Expected Deaths, and you will see there for Asbestos, SMR one point 0 two...that is the figure you have just quoted to me, and Thetford Mines, SMR one point one 0.

Now, if you look at that, you'll see in the quinquennia from 1926 through to 1975, the observed deaths and the expected deaths, and the excess is shown in black, okay? So you will see in Asbestos that it looks now as if we have worked through the excess period in Asbestos. In fact, I would expect quite...I wouldn't expect...I would not be surprised if thereafter... and of course, we have to bear this in mind, that if you have a lot of people dying early from one cause, they aren't allowed to die later from something else. So we would expect in a situation like this, unless there were still very long-term hazards at work, very long-term, if anything for this cohort now to have lower mortality than expected, and indeed we do see in 1971 to 1975 in Asbestos that we have a deficiency of deaths. Right? But in Thetford Mines, however, you will see that we are still in the black. I mean there is still an excess mortality still apparent in 1971, but I think it's probably meaningful that in 1971 to 1975 the excess is now diminishing. This would, I think, be reasonable in relation to what we know about the general latency of asbestos-related diseases, that the maximum risk probably is in the period of around thirty to forty years, say, and that having worked through that quite possibly we will now have less excess.

So the answer to your question really is, no, I would not expect us to find now a greater amount.

The only reservation I have on that, and I did

A. (cont'd.) say it in one of these papers somewhere, was that I would keep an open mind still on mesothelioma. But I don't think that will affect the issue very much, numerically, because the total contribution of mesothelioma is numerically small. But it is possible that we will go on having perhaps relatively more mesotheliomas the next five or ten years.

Q. I'm going to come to mesothelioma in some detail, but just stopping there for a moment, is that because of a longer latency period with respect to mesothelioma, or is that one of the possible reasons?

A. Yes, that would be where I would want to be a bit cautious. We do know that the latent period of mesothelioma is certainly, in this group, long. Again, we've graphed it here in that same paper, if I can refer to page seven, figure three of that paper shows, if you just for the moment look at the cases of mesothelioma in Time from First Exposure, and you see that those cases range from twenty-one years through to fifty...whatever it is...four or five years, and there is certainly nothing to suggest there that we necessarily reached the end of that little bunch of cases. It might well be that they will go on at the similar intensity for a few more years yet.

Q. The comparison in figure three, I take it, is with the gas mask workers study?

A. That's right.

Q. Of yours?

A. That's right. That's a bigger question.

Q. Can I come back to tab eighteen, and just at the bottom paragraph of the abstract, I take it another of your main conclusions is there cited, that if the only subjects studied had been the nineteen hundred and four men with at least twenty years employment in the lower dust concentrations averaging six point six million particles per cubic foot, or about twenty fibers per c.c., excess mortality would not have been

Q. (cont'd.) considered statistically significant except for pneumoconiosis.

Now, I just want to make sure that I have got the right tables by which you made that calculation, and can I first of all take you to page sixteen, table four, which I take it is a table which shows duration of employment correlated with dust exposure? Now, are the nineteen hundred and...

A. Table four, excuse me?

Q. Table four on page sixteen.

A. Yes, yes.

Q. Are the nineteen hundred and our persons the persons who appear in amongst those with length of service greater than twenty years in the low and medium dust exposures? That is, the one thousand and thirty-seven and the eight hundred and sixty-seven?

A. They should all be in the..yes, I mean they are certainly in that piece of more than twenty years, and I assume that it must...I don't know if that adds up to anywhere around nineteen hundred and four. If it does, we're lucky.

Q. I think it does.

A. Yes. Yes, it looks as if it does, it is those two groups.

Q. Can I then take it at page eighteen, table seven D, I take it this is your table on Death by Cause in Relation to Dust Concentration?

A. Right.

Q. Then do we then look to the SMR for lung cancer for the low and medium dust exposures? That is, the figures one point two one and one point zero eight?

A. Yes

Q. Then are those the two figures which, on which you base the conclusion that excess mortality from lung cancer is not statistically significant?

A. On those.

Q. On those two figures?

A. I think it must be those, yes.

Q. And that analysis was based on a twenty-year latency period?

A. All of them are.

Q. All of them are?

A. All of them are based upon twenty more years from first employment.

Q. Is there any particular reason why twenty was chosen? For example, we had Mr. Berry last week, and I gather his analyses are based on ten. Is there any current thinking on whether ten or twenty is a more appropriate figure?

A. No. I think not. I think Berry chose ten years because it was convenient. I think we have taken twenty in this because in earlier analyses...people have been criticized for not having a long enough one, because you...if you count the deaths before twenty years, in effect what you are really saying is that probably most deaths within twenty years or within X years of first employment are nothing to do with the job...if we are considering cancer or asbestosis...and therefore, any deaths...if you include shorter periods, you will be, to that extent, diluting the results. Dr. Selikoff has been extremely insistent that studies of asbestos workers should be at least twenty years. In fact, he has recently said they should, if possible, be thirty or more years. And we wanted to be sure that we could not be criticized for, if you like, picking a short interval.

It would make very little difference. We may, in other analyses, could demonstrate that it makes next to no difference, but that's the reason.

Before you leave that subject, however, you did quote to me something which I believe is from the abstract and which I think the first sentence of that last paragraph is a

A. (cont'd.) preamble to the last sentence. So in a sense, it is quoted out of context.

You see what we are really saying is, that while it is true that persons with this, below this level of exposure would not have...we would not have been able to say that the excess was statistically significant by sort of usual methods. We go on to say the inability of such a large study to detect increased risk at what would be quite unacceptable levels, points to the need for examining exposure-response models. Because, you see, our conclusion is not that there is no risk. I want to emphasize that. Our conclusion is that there is a risk, but that we can't demonstrate it, and that therefore the great importance of deciding what is the nature of the exposure-response model, and what we conclude from this is that the exposure-response model that fits best is a linear relationship, and that therefore, with a linear relationship we must assume that there is an excess mortality attributable to exposure at lower levels.

I want to make that clear because it could otherwise be read to imply that there is no hazard below twenty, and the reverse is our conclusion.

Q. I'm glad you did make that clear.

So I just want to make sure I understand that. Is what you are saying that epidemiological methods are not sensitive enough to detect the excess risk at low levels?

A. That's right.

Q. And therefore your data does fit a linear relationship, and I take it a linear relationship gives you some margin of safety, to some extent?

A. No, it doesn't.

Q. Not necessarily?

A. No. No, it...there are other models which could give you a bigger margin of safety. I mean, you could have

A. (cont'd.) a model which says that the exposure increases more rapidly at low exposures than it does later, and that would be a model which would imply a greater level of safety, or rather shall we say it would imply the risk of greater at low levels.

No, I think it is a model which ...it certainly gives you more safety than the old threshold model, although it is not...you can have a linear relationship which still has a threshold.

However, we are getting into another subject, perhaps.

Q. Yes. Your linear model...

A. Goes to zero.

Q. Goes to zero?

A. Yes.

Q. Then is the second, is another conclusion that follows from all of this then that you will then, to detect risk at low levels, will extrapolate along your linear relationship?

A. That's right. And that is our conclusion, if you want us to estimate risk at low levels, not our failure to detect a statistically significant level at twenty fibers.

Q. What figure would have been, or are you able to say what figure would have been statistically significant?

A. No. It's possible my statistical colleague might be willing to say something on that. I couldn't.

Q. Coming back to page eighteen, are the SMR's for pneumoconiosis or asbestosis, are they significant at all levels in workers with more than twenty years gross service?

A. I'm sure they are. Because I think your expectation...I mean it really, almost again, outside the statistical argument, your expectation of death from pneumoconiosis in persons not exposed to dust is zero, is very low.

I mean you have got your erroneous diagnosis,

A. (cont'd.) that's all.

Q. One, just one methodological question about table seven. I think I read in the text that you excluded in calculating deaths in table seven, all those of those deaths that occurred before 1951?

A. Yes.

Q. Could you just explain to us the thinking behind that?

A. Two reasons. There are no...there were no appropriate age-specific death rates for Quebec to give us an adequate expectation before that. We could have manufactured some, but we didn't have them.

The other reason for doing it, two other reasons, something like three-quarters of our data are still available, and they are the ones that are probably the best, because after all before 1950, things like diagnosis of lung cancer would have been more questionable than it was more recently.

Then finally, we did have to pick...we were very anxious to include data on smoking, and we decided that we couldn't go back before 1950 to get any sort of histories of smoking. So that was the reason.

In other analyses, we have gone back before 1950, if they are required.

Q. Is there likely to be any bias one way or the other from the exclusion of those deaths?

A. No.

Q. No?

A. No, not at all.

Q. One other matter I wondered about was the breakdown between low, medium, high and very high exposures, and then, as I understood it, attributing different figures to those categories, depending upon years of service. Can you just help me there with the thinking behind that approach?

Q. (cont'd.) I suppose as compared to simply taking one cumulative dust exposure figure, regardless of number of years?

A. I'm not sure that I entirely follow you. What we are doing here is to divide...what we are trying to do is to separate duration and concentration. Therefore, under table seven we have got the analyses in four categories. Now, I'm not sure if your question is why is it that we use the terms low, medium and high, very high, and that those terms don't mean exactly the same thing in the four tables? Is that what you mean?

Q. Essentially, as I read the text at the bottom of page thirteen and the top of page fourteen, low, medium and high in short-exposure categories means one, three and ten million particles per cubic foot year. So then when it's medium service, it's...the corresponding figures are...

A. That's right.

Q. ...six, thirty and a hundred?

A. Are different, yes.

Now, that's quite true. The full extent of that is shown in table four...

Q. Right.

A. ...which does show that for each of those four categories, in each of the four durations, the average dust level varies.

So, for example, in low, if we take in turn less than one year, one to five years, five to twenty years, more than twenty years, the average concentration there is not too bad - two point six, two point five, two point five, four point two, etc. But the reason for doing it was that we decided that it was more useful to group people into four reasonably-large groups of persons in each duration category, than it was to have identical concentration classifications, because it doesn't really matter, you see. I mean, we are not

A. (cont'd.) doing very many comparisons across between low..shall we say, high concentrations and then comparing high concentrations in four duration groups. We are more interested, really, in concentration than we are in duration.

I say that because I think biologically it is probably more important to know what the effect of dust concentration is than duration of employment, and secondly, because administratively I don't know of any proposals, apart from Eastern Europe, where one has considered limiting exposures to carcinogens in terms of duration rather than concentration. All standards are stated in terms of concentration, though of course it could be argued that you might do just as well by approaching it on duration. And as I say, some Eastern European countries are seriously looking at that.

Q. I see. Do your data respond, show a response relationship not only in terms of concentration or cumulative dose, but also in terms of exposure, duration of exposure?

In other words, were you able to separate out the effects of those two factors?

A. Not with any confidence. Not with any confidence.

We are satisfied that we have separated out the effect of concentration. We are not satisfied that we can say for a given concentration what is the effect of duration.

Q. But you are satisfied that you can say, for a given...

A. Duration...

Q. Duration.

A. ...what's the effect of concentration. Satisfied is a relative term. Relatively satisfied, yes.

Q. I see. Are there any tables in this paper that make that clear?

A. I don't recall one that does. It is

A. (cont'd.) possible that, not in this paper but in a paper which you may have, 1977 paper, the Royal Statistical Society Paper...

Q. I think that's tab thirteen.

A. Thirteen, that's right.

It is possible that in there somewhere this has been done. I'm sorry, I would need to look at it, if you want me to.

Q. Perhaps we can do that at the break, rather than...

A. I can't really remember the answer to that question.

I should, if it's relevant to you again, point out that one of the statisticians at McGill, the one associated here, Dr. Duncan Thomas, has been doing a great deal of work on this in recent years and might have some comments to make.

Q. Separating out the effect of those two factors?

A. Yes, yes. Amongst others, yes. You see, there are much more important factors even than that. There are issues such as the pattern of the exposure. You know, all the studies here are really averaging. They are not making any distinction here between, shall we say, a man who had a very high concentration at the beginning of this period and then a low one, as opposed to a low one and then a high one, or a series of fluctuating concentrations. You can make biological sort of...you can make hypotheses that it might make a difference and he has been attempting to look at this sort of situation to see if there is any evidence of different patterns of exposure having different effects. I think it's stretching the data a bit far, but that's what he is doing.

Q. I wanted to ask you, actually, about that very point. If I can take you to, again at tab eighteen, page fourteen.

Q. (cont'd.) Sorry. Again, at page fourteen in the first full paragraph. The paragraph starting 'relative risks'?

A. Yes.

Q. "The relative risks of lung cancer were considered in detail by Widdell, et al, and it appeared that there was little to suggest that the way in which dust exposure had been accumulated played any part in determining the risk".

Is that the kind of issue that you are talking about?

A. That is precisely what I was looking for, yes. That is what I wanted to find. I had a feeling we had said something on those lines somewhere. That did mean that they had looked to see if they could find any evidence that the pattern of accumulation of dust made a difference.

Q. Does that mean that the conclusion from their findings is that in fact intensity of exposure, at least insofar as they were able to determine, didn't appear to affect relative risk? That is, whether you've got short, intense bursts as opposed to less intense bursts but over a longer period of time?

A. That's right. I think that's what it comes to. I mean, the implication of this statement is also that duration is equivalent to concentration. In other words, that if you double the concentration, it is equivalent to half the...you know what I mean...you can get the same answer by either increasing the concentration or increasing the duration, as long as you halve the other one.

Q. Right.

A. The implication here is that they are equivalent. But I don't think...I think we should be very clear that this doesn't mean that there is evidence that this is so. It is rather that epidemiological information...and I don't want to downgrade this stuff, because it's more detailed than many...it nevertheless is terribly crude and inadequate for examining these really very refined points.

Q. Just one further question on that issue. I note from some of your papers that Mr. Berry fitted certain lines to your data.

A. Yes.

Q. Bearing in mind the testimony that he gave last week on differing ways of measuring dose, I am just wondering whether there was any consideration in your own study to differing ways of measuring dose?

A. Well, I mean there are two main ways, of course. One is dust and one is fiber. But I take it you don't mean that.

Q. No. I meant more his...

A. More a matter of do you look at particularly a model of dust which gives more weight to dust the longer it has been potentially in the lung.

Q. Yes.

A. Time weighted.

Q. With various clearance factors.

A. That's right. Well, as a matter of fact we have looked at that. I can't recall whether we've looked at it in mortality. We've certainly looked at it on radiological surveys. I would need, again, to look this point up, but I can tell you the problem, and that is that in a work force like this, whichever way you do it, all these indices are very highly correlated, and you can't really separate out which ...they all give the same answer. That's what I'm really getting at.

Q. They will all generally fit the data equally well?

A. They will all fit it. They will all fit it, because they are the same man, and whichever way you do it, there are very high correlations between...I think we used six different indices of exposure, including some that took into account work stress, physical effort required, and at the end of the day you

A. (cont'd.) have indices which are very highly correlated and you can't separate out the...and if one did give you a better answer than the other, it might be due to chance.

Q. Thanks, Dr. McDonald.

I take it from, again back in your paper at tab eighteen, that from table eight, which is at the top of page nineteen, you basically reached generally the same result approaching it in a slightly different way.

Can you help us as to the thinking behind this other approach, which I gather was to measure accumulated dust exposure to age forty-five?

A. Yeah. There is a very serious problem in all occupational studies of chronic disease, that if we imagine that a measure of exposure has got a time factor in it, that we are not only considering concentration but duration, and while I have just now said that it is very difficult to kind of put a value on the duration part, I don't think that there is any reasonable belief that duration hasn't got anything to do with it. In other words, that exposure at a certain concentration for a day almost on common sense grounds has to be less hazardous than exposure at that concentration for thirty years.

So duration must have something to do with it, and therefore any form of index of exposure in occupational studies has to, to some extent, take into account both concentration and duration. The way we've done it, and it's a way in which others have that have done this sort of thing, is to simply multiply one by the other, in effect.

Now you see that the net effect of that is that it becomes..in order to accumulate a high exposure, you have therefore got to work a long time, and you've therefore got to survive a long time.

It follows therefore, that if...I mean, one way would you say how do you determine the effects of a lifetime's exposure to asbestos? You might say that the answer was to take

A. (cont'd.) people who worked for a lifetime in asbestos and look to their mortality experience.

The snag with that is that that would automatically sort out the ones that had been able to work for a lifetime, and had not either left or died in the meantime.

So you have this problem that in order to accumulate a long exposure you've got to survive.

So the question is, what can you do about it? The classical example of a situation in which...I can give you two classical examples, one from Asbestos...in which you really want to separate completely the acquisition of exposure from the measurement of effect, so that one doesn't contaminate the other. The ideal, if you like, the classical situation, is an atomic bomb which drops, everybody gets whatever exposure they are going to get, and then you watch for the next fifty years to see what will happen to them. There is no contamination of one or the other, and you will see that we approach that in the men who made gas masks. They were exposed from anything from a few months to a maximum of two and a half years, and their effects were totally separated later. Duration was not relating, therefore, to survival from the disease you are concerned about.

Okay, so...it's a long answer to saying why we did this. We said, right, in practice most people get a very high proportion of all the exposure they are going to get by age forty-five, especially in an industry where the dust exposure is improving. Anything that happens after forty-five is quite small, whereas a very high proportion of all the deaths we are interested in occur after forty-five, and therefore by making this arbitrary line we say we are classified by people up to forty-five and measure the deaths after forty-five.

Q. I see.

A. Didn't get a different answer though.

Q. Can I take you back to tab seventeen, your

Q. (cont'd.) article in the Annals of the New York Academy of Sciences?

A. Yes.

Q. There was one question about the linearity of dose-response relationships which I wanted to ask you about, and it appears at the top of page eight. You there talk about reporting that your relationships may well be linear, and that linear relationships are attractive for legislative and control purposes because once assumed, they permit calculation of relative risk at low exposure levels, as you have indicated previously.

Then you go on to say, "For malignant disease, linear relationships are biologically plausible enough, but less so when applied to deaths from pulmonary fibrosis."

I wonder if you could elaborate on that statement?

A. First of all, the plausibility in relation to malignant disease. If we imagine that at least one component, and I now speak without any knowledge of what I'm talking about, if we imagine that one component of the onset of malignant disease is damage to the nucleus to produce a mutation, each insult to the nucleus of cells, in this case lung cells, will carry, if you like, a finite risk that something will go wrong. So it is reasonable that the dose should be directly related to the probability of this mutation occurring.

This will hold up even if there is a, if you like, a repair mechanism, because the repair mechanism will still, if you like, have to be dealing with a hazard which is dose-related. As far as I can see, it cannot easily turn the relationship from a direct linear one to any other form.

I may be quite wrong and toxicologists may know much more about it, and geneticists, much more about it than I do.

Okay, so I don't have any great difficulty in

A. (cont'd.) accepting that that sort of mechanism is reasonable for cancer.

But if we start thinking of pneumoconiosis, it doesn't seem to me impossible again that there may be some sort of finite relationship between one fiber of asbestosis and a probability of a little bit of fibrosis occurring in the lungs. But we haven't got now a process in which, if you like, once set off, one little bit of fibrosis will progress until it kills you, like in cancer. We have a situation in which that fibrosis has got to accumulate and accumulate, and indeed it doesn't really kill you direct anyway. It mostly kills by eventually so embarrassing the function of the lung or the function of the heart that eventually the person succumbs from something else.

I find that sort of process much more difficult to relate directly to...I think, it would seem to me you've got to have fibrosis beyond a certain level before it could conceivably do anything to you, because the reserve power of the lung is fairly substantial, and it seems to me you've got to make some significant reduction in your lung capacity before it could really kill or lead to increased probability of death.

Having said that, we still find a linear relationship.

Q. For both asbestosis and lung cancer?

A. Yes, and for, almost for gastrointestinal cancer, too. Rather less good, but still there.

Q. But not for cancer of the larynx?

A. We don't find any excessive cancer of the larynx in the Quebec mines.

Q. One of...one proposition that was put to us last week by Dr. Weill was the proposition that a fibrogenic dose is also a carcinogenic dose, and I note that you have some comment on...I think you have some comment on that point in your article which is at tab twenty, Radiological Findings as Predicters

Q. (cont'd.) of Mortality in Quebec Asbestos Workers.

I take you to page 266, and at the first full paragraph at the top you say, "The effects of asbestos are fibrogenic and carcinogenic. In pathological terms these responses are distinct and will both appear to be directly related to exposure. They may or may not occur independently."

Then carrying on at the top of the next page, the top of the next column, you say, "The nature of the association between pulmonary fibrosis and lung cancer in asbestos workers is a matter of theoretical interest and medicolegal importance which our findings do not wholly clarify".

Does that mean that your findings have not found any medical or biological association between the two?

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Edwina Macht

EDWINA MACHT

...to page 36

A. I think, perhaps, it's more a matter, it seems to me, that studies of this kind probably can't answer the question. We aren't aware where the problem lies.

I think the problem lies, first, on what you mean by pulmonary fibrosis, because, certainly, if we are looking at radiological evidence, there is certainly no assurance that the X-ray is a perfect one-to-one detector of pulmonary fibrosis; it's a way, way from that.

It is very possible that there is pulmonary fibrosis without X-ray changes being read -- and let's make that clear -- it's a reading we're looking at; not any absolute, God-given change. It's a judgement by some readers.

And the reverse: that I'm sure there could be fibrosis read, or changes compatible with fibrosis read, and yet, at autopsy, no fibrosis. Again, the fibrosis would depend at what level you're looking at it. I mean, how detailed an autopsy or biopsy examination were done to find out whether there were any fibrosis.

So we're looking at much too crude a measure, anyway.

I think -- and I would need to look back at this paper, but if I remember exactly, what we find is evidence that is compatible.

You see, the next point I wanted to make was, on the one hand, there's that problem; the next is, how do you decide which of the many lung cancers are attributable to asbestos, and to what degree they're attributable to asbestos exposure, given that there is a normal expectation of lung cancer; it's quite a common cancer in males, anyway, who are not asbestos workers.

So we can't identify the ones that are asbestos cancers, even if we knew everything in order to be able to say,

A. (cont'd.) is there fibrosis in all of those? We don't know that.

And so, I suppose the quick answer is, I don't think our data does -- it is compatible with most excess lung cancers being in people who had X-ray abnormality, but there's also evidence in our data of some excess lung cancer not having been explained in that way; okay?

And I suppose, really, the argument I would make would be very much more a theoretical one here. The argument would be that I think there is no special reason to believe that the two processes of fibrogenesis (that is, asbestosis and lung cancer) are the same mechanism. They are different mechanisms, apparently.

They are both, apparently, exposure-related, but it seems to be no reason to believe that the factors of susceptibility will be identical for both; and, if they were identical for both, then we would expect, anybody who shows fibrosis to have a finite risk of getting lung cancer. And then, all lung cancers would be in people who had some fibrosis; right?

But I would advance the point that that's an unreasonable view; that it is quite possible that a few -- maybe not many -- but that some people are susceptible to lung cancer but are not susceptible to fibrosis, and do not demonstrate any important degree of fibrosis, and yet, conceivably, have had their risk of lung cancer increased by asbestos.

So, I mean, that certainly doesn't help the medicolegal problem, because I think the medicolegal problem is enormously difficult.

Q. When you say the medicolegal problem, I take it you mean the problem **at** the **individ**ual level ---

A. Attributability.

Q. --- of compensation and the question of

Q. (cont'd.) attributability?

A. That's right; that's right.

Q. And do I take it from your comments that you're suggesting it may not be reasonable simply to compensate for lung cancer only those persons who also have some evidence of asbestosis?

A. I think that that -- I think that the issue of compensation is really a matter which you cannot extrapolate in any disease directly from the scientific evidence. I mean, I doubt if the scientific evidence is ever adequate, anyway, but I think that, at some point or other, what is involved is a social judgement.

I think that -- all I'm saying is that, theoretically, I would not like to say, categorically, that a case attributable to asbestosis has to have fibrosis, but, having said that, I think it's probably a very small probability that somebody will have enough exposure to asbestos to give him lung cancer, and yet have no evidence whatever of fibrosis.

But, you see, a lot comes down to, what do we mean by the evidence of fibrosis? I would think we probably could find some evidence of fibrosis, if we looked hard enough, in most people in this room.

Q. Do you, yourself, have any professional opinion on that; what is fibrosis, what is asbestosis?

A. I don't have any difficulty about asbestosis, because I think asbestosis is a disease, so I think you would have to have some, from my way of thinking, something which was recognizably a disease; that is, something which was actually causing either symptoms or function impairment. I think it is more than a shadow on an X-ray, or more than a few -- a certain number of cells of a certain type in the lung to have a disease.

But if we mean, is there evidence of fibrosis,

A. (cont'd.) pathologically, then I mean -- I think this is where I get outside my area, but I'm sure some pathologists would say there is evidence of fibrosis there. Then they would have the problem, is that fibrosis due to asbestos, because quite a high -- there's probably people who could answer it better than I can -- but I believe that, in industrial cities, a high proportion of persons, my age and over, have got some fibrosis at death.

Q. Can I just explore the relationship, or lack of relationship, between fibrosis and lung cancer with one more question.

Dr. Weill's testimony explored it not only from the individual point of view but from the standard-setting point of view, and, as I understood his submission, it was to the effect that, if you set a standard sufficient to protect against excess risk of asbestosis, you will likely be setting a standard that will protect against excess risk of lung cancer.

And I guess my question to you is whether your own research into the Quebec miners and millers cast any light on that proposition. Can you draw that conclusion from your own work?

A. I don't think we could, but I think that I certainly want to include in the argument mesothelioma. I say that because, after all, some mesotheliomas -- it's not fair to call it a lung cancer, but it's a respiratory cancer of sorts (or some of them are), and I certainly would say that exposures to asbestos have produced mesotheliomas without producing detectable fibrosis.

But then, you see, we haven't got on to the issues of things like types of fibre which could have an effect here.

MR. LASKIN: I wonder, Mr. Chairman, if you might want to take a short break.

DR. DUPRE: Certainly. Shall we reconvene at quarter to 12:00?

MR. LASKIN: Sure.

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DR. DUPRE: Counsel, are you ready?

MR. LASKIN: I am, Mr. Chairman.

Q. Dr. McDonald, just one final question on the matter we were dealing with just before the recess.

Leaving aside mesothelioma for the moment, does your research cast any light on whether a standard low enough to protect against excess risk for asbestosis will also be a standard low enough to protect against excess risk for lung cancer?

THE WITNESS: A. You gather, from my silence, that it clearly doesn't, so easily. What I think we know is that, first of all, by any standard, there is a lot more asbestosis than there is lung cancer, so that, in this cohort of ours, we estimated that, I think, forty-six or fifty deaths from lung cancer were attributable to their work in this large cohort. It's not very many; it's about one-fifth of all the lung cancers that occur.

On the other hand, depending on your definition of asbestosis, but even on grounds of, you know, if that same cohort had completed a life's work, some of them, it can be debated what proportion would have radiological evidence -- definite radiological evidence of asbestosis. Not necessarily any serious disability, but radiological changes which most people would say were abnormal. Maybe our figure is around about a third would. Others will find higher rates than that;

A. (cont'd.) okay?

So we're going to have more asbestosis, and we do know that both asbestosis (radiologically, functionally, and symptomatically) is dose-related, much less clearly than the lung cancer is, but dose-related. Now, where does that put us?

It means that, certainly, on the face of it, there's an awful lot more asbestosis to prevent than there is lung cancer, and it therefore seems to me likely that, in practical terms, if you could reduce your asbestosis to negligible quantities, almost for certain you would reduce your lung cancer to undetectable, because there's an awful lot more of the asbestosis.

And so it seems to me reasonable that, if you could get the asbestosis down, the lung cancer would no longer be detectable; and, therefore, for practical purposes, I think this is probably true. But I would simply put in the kind of theoretical rider that it still seems possible to me that there might be the odd case of lung cancer occurring, that you'd perhaps never be able to identify, which probably was, to a degree, attributable to the work in which there was no important amount of fibrosis.

Q. And you say there's lot of asbestosis around, do I take it you mean not only asbestosis where that's the primary cause of death, if you will, but also asbestosis which may be present but may have contributed to death by another cause?

A. Well, when I was saying it just now, I was referring to asbestosis as a manifestation in life; a man with some degree of mottling on his X-ray -- if your X-ray readers said, "Here's a man who worked for thirty years in the asbestos industry, and that's his X-ray, what do you think he's got?" I think people would say, "Yes, he's got abnormalities which are compatible with asbestos exposure." But he might well have no

A. (cont'd.) important disability or symptoms or diminished expectation of life.

Q. I see.

A. I meant it in that sense. If you mean, is there more asbestosis of the severity that will kill, then we've fewer deaths from that, from asbestosis, as such, than from lung cancer.

Now, I meant it in the usual sense of a disease diagnosable in life.

Q. Can I turn now to the question of fibre conversions, and I appreciate that Dr. Gibbs is the person who, I take it, is mainly doing this work, and we're going to have the privilege of having Dr. Gibbs, but I wonder if, in a general way, I might just ask you a few questions about it.

I take it, for some period of time, your group has been attempting to convert your dust particle measurements to fibre measurements, and do I also take it that that work is ongoing at the present time?

A. It's more or less fizzling out, actually. I mean, we're beginning to get depressed about it. [Laughter.] We've been doing it for a good five or six years, and I think we know how almost unanswerable the problem is.

Q. From reading your own papers, that's the message that seems to come through, and I gather perhaps the one reason that you attempted it was because legislators, for whatever reason, now express standards in terms of fibres rather than dust particles. Is that fair?

A. Yes; I think that's the dominating factor that, so long as you use only measures of dust, people will say, "Ah; but it's fibres that do you the damage."

And it is, I think, on theoretical grounds, reasonable to legislate in terms of fibres, because, presumably,

A. (cont'd.) it's the fibres that cause the disease; but, having said that, unfortunately we've got virtually no epidemiological data of any kind in the world which relates to fibre. I may be slightly exaggerating, but I can't think of one study, offhand.

So we're going to nearly always be in the situation of having to make some sort of conversion.

Q. Is it your -- perhaps I could just ask you, and would it be fair to ask you whether it's your professional judgement, in view of the comment that you just made, that we should be looking to set standards in terms of what we've been able to measure, dust particles, rather than what we haven't been able to measure: fibres?

A. I don't know. I suppose, speaking personally, my view about environmental standards is that I am a bit dubious about the concept of flat standards. I think that probably one will do better to relate -- so far as occupational exposure is concerned, to use the experience of a given industry to control its hazards, rather than -- after all, the concept of the fibre standard was, all exposures are really due to fibres; therefore, we can go to this common multiple that then can be applied in any situation; whereas, I think there's quite a lot of evidence now that, even if you converted to fibres, the fibre wouldn't mean the same thing in two different places.

Do you know what I mean; that a fibre count doesn't mean the same thing in a textile industry as it does in a mill.

Q. Can you elaborate on that.

A. Well, I'll elaborate on it mainly in the sense that we have got, clearly, exposure -- not so clearly, but such data as there are, the exposure-response relationships look quite different in different industries.

A. (cont'd.) I have some knowledge of this plant, recently, in South Carolina where NIOSH people have been doing a study, Dement, and others, which is a textile plant in which, in terms of fibre concentrations, the fibres apparently -- the concentrations are very low (I mean, very, very low), compared with anything in the Quebec mill.

And I see no particular reason to think they're wrong. They're, you know, sort of subject to some errors, but they have several -- one to two orders of magnitude different. Now, what -- how does one interpret the findings?

You can either say that there's something special about the textile industry which is much more hazardous -- nothing to do with asbestos, but some other thing. You know, you can't even dismiss that.

Q. Some co-carcinogen, some other ---

A. Spraying with oil is done in the textile industry. You know, there is this conceivable possibility. But I think it's unlikely; I think it more likely that -- I mean, something that has been more or less showing through all the asbestos information for twenty years now, and that is that it looks as if the further you take the fibre from the mine, the more it gets divided and the more able it is to cause disease, in general terms.

Q. And is that because, the more it gets divided, the finer it is, and therefore, presumably, the easier it is to get into the lung?

A. No, no; I think not. I think it is that the fine fibre -- again, this is not an epidemiological opinion, but I think the toxicologists would probably support the view that the fine fibres are more liable to cause both fibrogenic and cancerous changes in the cell.

From the point of view of penetration to the lung,

A. (cont'd.) I think we have a further factor; and that is the shape of the fibre being most important. And the shape, particularly in terms of length and curliness.

Q. And what shape more easily gets into the lung?

A. A small, straight one; a needle.

Q. And I take it that that comment is directed more towards the issue of fibre type, or is it also directed towards the question of ---

A. Well, you could say, regardless of what it's made of, that a straight, small needle will get in better than a curly thing.

Q. Is there some indication, or is there some evidence, that, as you get farther away from the mine, that the fibre is also changing shape?

A. Not shape; not so much shape, as thickness.

Q. As thickness?

A. Yes. And I think it's the thickness side of the thing, the thickness plus length, that is related to disease-producing quality, and partly the dimensions and the configuration (the curliness and so on), which affects ability to penetrate the air passages. So you've got two factors going on, at least.

MR. LASKIN: I think Dr. Uffen had a ---

DR. UFFEN: I have a related one here.

Could it have anything to do with the ability of the natural-occurring fibre to cleave; whereas, man-made fibres might be glassy, and not cleave?

THE WITNESS: You mean, split -- divide longitudinally?

DR. UFFEN: Yes.

THE WITNESS: Yes, definitely; definitely. I mean, this is one of the characteristics of asbestos, that it --

THE WITNESS: (cont'd.) I see some there -- if you damage it, it doesn't tend to break longitudinally; it tends to break across.

DR. UFFEN: Does it ever happen within the lungs, after it has got there?

THE WITNESS: I don't think we can answer that question; I'm not sure whether the animal experimenters could answer your question. They would be the only people, I think, who might be able to.

And I think one of the main problems in that connection, incidentally, is that the animal studies have a serious limitation in duration of time, because just the sheer length of life of animals, in relation to a physical process, like, for example, dividing up, might be really different from that of man, where twenty, thirty, forty years is involved; I don't know.

MR. LASKIN: Q. Then coming back to the question we were discussing, and your concern about setting standards in terms of a particular level, could you help me a little further on what you meant by the industry -- looking at particular industries; are you suggesting that different standards, perhaps, should be set for different phases of the asbestos industry?

THE WITNESS: A. I'm rather reluctant to get into a discussion of standards at all, to tell you the truth, because I don't really think standard-setting is a job for people in scientific work.

I also think that I don't believe in making the question -- it's a very difficult question, and I think I would try to simplify it, so I wouldn't -- I mean, I would say there are other grounds, at present, for saying that we probably would be wise not to use any amphibole asbestos, so that we are only

A. (cont'd.) dealing with chrysotile. And then I think probably we would be wise to do what the industry already is doing, in fact, and that is, look only, for all intents and purposes, at friction and cement products.

Now, if you get down to that, and that must be a very high proportion of all current use of asbestos, it leaves only real problem, actually, which needs to be looked at, and that is, how can you make large pipes without the amphiboles? But I'm told this can be done, if you want to.

If you then get down to that and say, okay, we're only dealing with the problem of asbestos cement and friction products, it seems to me that it should be possible for those two industries to examine the epidemiological data for those two industries and start making intelligent application of that information.

Q. Then, just following it up -- and I don't intend to get you into an area of social policy, and so on, but, from the epidemiological point of view, is the approach in setting a standard, whatever it may be, on the basis of a time-weighted average per particular eight-hour day, or whatever; are you suggesting that that may not be an appropriate approach; that there's some other standard, some other way of fixing a standard that we should be looking at?

A. No, no; I don't find anything terribly wrong with this. I think one of the sort of complications that's beginning to creep into the picture is that our occupational hygiene, quite rightly, say that the area sample is not necessarily reflecting what individual workers get, and, therefore, they will then be saying that "What we want to do is to control the, if you like -- some form of personal sampling," which is rational, up to a point, and useful for what you might call "in-factory control measures."

A. (cont'd.) But I think perhaps it's sometimes forgotten that, if there is no data on fibre concentration, there's even less than no data of an epidemiological nature which relates to personal sampling; there is none there. And we, therefore, have got nothing to go on on personal sampling, but that doesn't mean that I would say that it isn't useful to do some of that; no.

I think it's perfectly reasonable to base environmental conditions on sampling of the working place, and, subject to certain assumptions regarding the length of the working day, and so on, to make some sort of control limit on that.

Q. And coming back to the question that I started with, is it sensible, then, to measure it in terms of fibres or, given our current knowledge, more sensible in terms of dust particles?

A. Oh, no -- I mean, I think it will depend on the industry. I would think that, in the asbestos cement industry, it would probably not be very sensible to control in terms of dust, because most of the dust will not be asbestos. That's true of the mining and milling, too; but, shall we say, in making absestos cement, you clearly are using a relatively small amount of absestos, mixing in with an awful lot of dust. And to just control on dust would probably be very misleading.

I would think that, in that industry, almost for sure you'd need look, primarily, at fibres. But I think that, say, mining and milling could make a very strong case for saying that we could control our industry very well on dust sampling, because there probably is a reasonably constant relationship (even although it's difficult to define) -- a reasonably constant relationship between fibre to dust in the mills.

And that certainly we know -- we've got epidemiological data which would help us to do it; whereas, if we do it

A. (cont'd.) on fibre, we probably are getting further away into an area of more uncertainty than we are into greater certainty; that's all.

But I think it's easy to say that sort of thing. I think you would have to be putting the clock back at least ten years to say that. I don't think that's a feasible approach any more.

MR. LASKIN: Does the Chairman have a question? I didn't ...

DR. DUPRE: Dr. McDonald, I do want to pursue this, just to make certain I understand your answer.

As I listened to your answer, my mind was going back to your 1974 paper, which is under tab 11, at page 68.

THE WITNESS: 68?

DR. DUPRE: 68. And you express an opinion there, in the words that begin at the top: "Though safety standards expressed ..." et cetera.

THE WITNESS: Yes.

DR. DUPRE: I was reading those words, as I was listening to your answer, and can I take it that your opinion, basically, has not changed from what it was in 1974, save that, today, you did single out the cement industry as one facet of the overall asbestos industry in which a fibre count rather than a dust particle count would be perhaps a more reasonable way to establish a standard, if you were going to have one?

THE WITNESS: Yes, sir; I think that's probably true. I think that, on page 68, you will note that we do say:

> "Our studies appear to provide a reasonable basis for establishing safety standards for chrysotile mining and milling ..."

This paper is very specifically concerned with chrysotile mining and milling and not concerned with any other

THE WITNESS: (cont'd.) industry whatever. And if -- so far as chrysotile mining and milling is concerned, I believe that the mining and milling industry could achieve control of its health levels by continuation of dust measurement perfectly well; indeed, probably with much less argument than will be brought up by fibre measurement.

But if we then look at other kinds of industry, I would -- you know, that wouldn't necessary apply at all, and I mentioned this other -- it certainly wouldn't apply for textiles, for example. For reasons of simplicity, I thought, let's try and get rid of the textiles; I imagine we haven't got to legislate for them. But if we did have to legislate for them, then I can't say how you could try to legislate for textile industry in terms of dust; you'd have to consider fibres.

DR. DUPRE: But could I read you the sentence that immediately follows the one you just quoted. This is your '74 paper again:

> "Without more evidence on the conversion factor that should be applied in different parts of the industry ..."

And I take it, parts different from mining and milling.

THE WITNESS: No, sir. "Of the industry"; the industry is the mining and milling industry.

DR. DUPRE: Oh; of the mining and milling industry; okay. I understand. Thank you, Dr. McDonald.

MR. LASKIN: I believe Dr. Uffen has a question.

DR. UFFEN: Could I follow up. Obviously we're interested in this conversion business, and I'm hopping back to the tab 17 again, the paper we've been discussing, and, on page 8, at the bottom of the page, there's a paragraph that discusses your colleague's work -- Dr. Gibbs --

THE WITNESS: Sorry; could you -- which paper is

THE WITNESS: (cont'd.) this again; the -- 17, page 8.

MR. LASKIN: It relates to the evidence before the Beaudry Commission on conversion factors.

THE WITNESS: Yes.

DR. UFFEN: It draws attention to the fact, there, that somebody analyzed your colleague's data and suggested a curvilinear relationship with conversion factors at relative dust levels, ranging from three to seven fibre per millilitre for each million parts per cubic foot.

THE WITNESS: Yes.

DR. UFFEN: Does this mean that a linear dose-response curve, based on particles, might become a curved dose-response curve when converted to fibres?

THE WITNESS: I think it's conceivable. It doesn't, in fact.

DR. UFFEN: It doesn't; okay.

THE WITNESS: It doesn't, in fact, because we've got another paper on this. We have, in fact, converted our mortality dose-response to fibres.

DR. UFFEN: And they stayed linear?

THE WITNESS: They stayed linear. I say that with some trepidation, because, I mean, in the end, we did what we say we really can't do; we, in the end, have been forced -- can I -- it's a long answer -- longer answer than that -- can I just say that, in the light of the data available to us in the early to mid-1970's, we were very depressed about the ability to convert from particles to fibres with any single conversion factor.

As is stated here, this analysis done for the Beaudry Commission gave a range from three to seven. Well, in fact, it was much bigger than three to seven; it was from about point one to about forty. And the curvilinear thing depended on

THE WITNESS: (cont'd.) what the -- at different dust levels, you get a different conversion factor; and the three to seven applies to the sort of levels we were concerned with; okay.

So, depending on what is the range of exposures that you're considering, so you would get different conversion factors; but, over and above the general pattern of range, there is enormous variation from place to place, from job to job, from mine to mine, and mill to mill; okay.

So what we decided to do at that point was say, "We can't convert" -- I was particularly influenced by Dr. Gibbs in this -- I say this in a sort of humorous way, because I think the environmental engineer is much more concerned with precision than epidemiologists are. I was not so worried about the poor conversion as he was.

I mean, if you try reading X-rays, you realize that precision is quite relative. So, okay; he was quite adamant, you couldn't convert them. So we said, "All right; we will now move and we'll look at all our data again, and instead of converting anything, you, Dr. Gibbs, will get these jobs and you will estimate, from first principles, what their fibre exposure was. No conversion about it; use the data you have and try to estimate what the fibre concentrations were."

Well, he'll be able to tell you that he gave that up after a while. He found he really couldn't do it. And we had to fall back on what was more or less -- it was less than a halfway house -- we did, in fact, use conversion. We did use not a flat rate of conversion, but we -- what, in effect, he did was to determine conversions for different parts of the industry for different jobs.

And, on the basis of that, we converted our exposures, which were expressed in particles, to fibres; but it wasn't done as I believe it theoretically should be done.

THE WITNESS: (cont'd.) Theoretically, I think one should really estimate the exposures in fibres, and then see what the answer is.

So -- I say this, because, in the end, we do still our same linear sort of relationship, but that could perhaps be due to the fact that a very high proportion of what we were doing was really still converting -- converting with a rather limited number of conversion factors. So we really were getting the same pattern again.

But what, at least, we were able to do is to say, "All right; with all the limitations we've now stated, very roughly, this is the -- instead of having the scale in terms of particles, we can now present our best available estimate of what that scale looks like in fibre concentrations."

MR. LASKIN: Q. I wonder if I can take you just through that process.

I take it there was an intermediate level when you wrote your 1980 paper, and I'm looking at page 21 of tab 18.

THE WITNESS: A. Page 21? Yes.

Q. And at the -- in the second column, at the end of the first paragraph there, there appears to be some indication that Dr. Gibbs or yourself -- you'd reconsidered your conversion factors and found they were probably in a lower range; between one and five, rather than three to seven.

A. Mmm-hmm. That's correct.

Q. And then, do I take it that the main paper in which -- in many recent papers, is the one at tab 21?

A. That's correct. There's one other, too, which I can give. This is the mortality.

Q. And then, if I can take you, first of all, to page 812 of tab 21, and under the heading "Field work," there's some indication that there had been some inconsistencies in work

Q. (cont'd.) histories and other evidence of possible errors. Can you just elaborate upon that a little bit. I take it there was a refining of the data, and a re-evaluating of your past data?

A. Yes, yes. You know, inevitably, over -- it was fourteen years, a number of inconsistencies had come to light in work histories, where a man was said to be employed by company A and you find that he wasn't employed by company A; that was his brother. And he was actually employed in some other company.

Or, indeed, industry will be interested to know how many people that are on their list that are not of their company at all. I mean, there are errors in work histories, and we became aware of them, as we -- in our tracing and cross-checking, and so on.

Q. I take it, from looking at one or two of your tables, there didn't appear to be any ---

A. It was very trivial.

Q. Trivial differences.

A. Very trivial.

One important factor we became aware of was, for example, in -- certainly in the Johns-Manville plant in Asbestos, it was the practice, when people went away on military service during the war, to keep them on the list of employed people, and, therefore, shall we say -- anything; one, two, three years, a man was recorded as working as an asbestos worker, when, in fact, he was in France, or somewhere. So this kind of thing we checked up on and removed a few years of exposure here and there. In practice, it made no difference, but we felt a little bit more secure.

Q. Now, coming, then, to the thrust of this paper, I take it, first of all, that this was a paper that dealt only with lung cancer, and was, in effect, a case control analysis; is

Q. (cont'd.) that correct?

A. Correct.

Q. And then, can I take you to page 814. I suppose, what -- am I correct that what was done here was, first of all -- Mr. Berry, who we heard from last week, fitted your data, your original data, based on dust particle measurements, to a line and got, presumably, a slope, whose equation, in terms of relative risk, is expressed on page 812?

A. That's correct; yes.

Q. Is that right?

A. Yes; yes, I think that's right. Yes.

Q. And then, do I take it you developed a conversion factor, an average conversion factor, between particles and fibres, which was 3.14?

A. Mmm-hmm; correct.

Q. And then applied that conversion factor to Mr. Berry's equation?

A. For the purposes of **this**, yes.

Q. And we then get, I take it, the equation that is expressed somewhere near the bottom of page 814, in terms of relative risk?

A. 814 -- yes; yes.

Q. And I suppose what we are interested in is the sentence, in broad terms and with many assumptions, the resulting equation which you then express would imply that the risk of lung cancer over a forty-year working life is increased by about 1.5 per cent with every fibre per millilitre concentration?

A. Yes.

Q. Can you help me as to, how do you get that percentage from the statistics?

A. I think it is by using the equation above and simply fitting into that equation a standard forty-year working

A. (cont'd.) life, and then working out what the -- using exactly that equation -- what the increments of risk are as you increase the fibre concentration. It's simply the direct application of that formula two lines above.

Q. And if we stick, then, only with lung cancer, and just with that equation, and I take it with all the assumptions that are built into your estimate, does the estimate then mean that, if we had a standard of two fibres in a work place, that the excess risk of lung cancer would be three per cent?

A. In asbestos mines and mills.

Q. In asbestos mines and mills.

A. Chrysotile asbestos mines and mills.

Can I add, sir, that we have now ---

Q. Please add anything else you want to.

A. --- this was, as it states here, a preliminary report, and we have since completed this study, so that we now have, and hopefully will publish before long, this analysis; but, for lung cancer, pneumoconiosis, gastrointestinal cancer, not with one control but with four, or three, depending on -- and mesothelioma -- and with a slightly more sophisticated type of analysis.

I merely say that because, yes, there is more data; this didn't use it all. And it doesn't make -- you know, this still stands up.

Q. And the estimate that you have there stands up very closely?

A. Very closely.

DR. UFFEN: Counsel, may I just ---

MR. LASKIN: By all means, Dr. Uffen.

DR. UFFEN: The 1.5 per cent for every fibre per millilitre concentration, that depends on the assumption that the conversion factor is linear, or constant; would I be right?

DR. UFFEN: (cont'd.) If you had a different conversion factor at a low level from what you used at a high level, then you couldn't give that single figure?

THE WITNESS: What you're saying is correct; what I can't remember now is whether we did it in this simple way in our further paper. I've got a feeling that we, in the further paper, have, in fact, applied conversion factors which vary. But, even so, it would still be subject to that limitation; that it would only be true to the extent that those conversion factors were the ones that applied. And if you were in a situation where they didn't apply, you'd get a different answer.

MR. LASKIN: Dr. Mustard.

DR. MUSTARD: Can I ask a question about tab 21, table 1, just to make sure I understand it correctly.

THE WITNESS: Which page is this?

DR. MUSTARD: Page 815.

THE WITNESS: Yes.

DR. MUSTARD: Because you're doing the studies the way you outline them at the beginning, you're determining the relative risk for this analysis based on the relationship between your cases and controls with people with exposures of less than thirty millions of particles per cubic foot?

THE WITNESS: That's right; yes.

DR. MUSTARD: So that, is it fair to say, then, that contains an assumption, does it not; that's not a true -- a sense of true relative risk; it's a relative risk based on the assumption that you're going to use that ratio as one so, in effect, if you actually do the calculation, the relative risk for the cases is less than one?

THE WITNESS: I think it's a true relative risk. It's a true relative risk -- well, it isn't; it's an absolute risk.

DR. MUSTARD: Well ... But it depends on what your controls are, doesn't it?

THE WITNESS: Well, okay; the question is, our relative risk is relative to these people; okay. It is relative.

So what would be -- I mean, in any relative risk, you've got to state what your basis is. I'm not sure what alternative you have available to you.

DR. MUSTARD: Oh; I quite appreciate that. But I'm simply saying, that ratio then becomes a determinate of a risk of one throughout the rest of your calculations; is that right?

THE WITNESS: Oh, yes; that's correct.

DR. MUSTARD: And that is somewhat different than some of the other ratios we've been seeing in some of the other papers. That was a point you made earlier, but I just thought I'd like to make certain I fully understood that.

THE WITNESS: Yes; this is true. I've forgotten which paper it is now; I think it is this 18 paper which shows that the relative risks obtained -- in the paper 18; that's our main mortality paper, I think the relative -- you see, you can obviously calculate your relative risks also from standardized mortality rates, and the -- and also you can calculate your standardized mortality rates -- we did it in two ways -- depending on all exposure, or exposure up to forty-five, or whatever it was.

And my recollection is that the relative risks in relation to dose didn't vary very greatly. I mean, in relation to the inherent error of the whole thing.

MR. LASKIN: Just one or two final questions on this issue of conversion.

Both Dr. Weill and Mr. Berry presented to us some

MR. LASKIN: (cont'd.) papers, which you may have seen, in which they each attempted conversions from dust particles to fibres in the particular phases of the asbestos industry which they were investigating.

Q. Is it a fair conclusion that the asbestos fibre content in dust particles seems to be less the farther you get away from the mine; or, at least, it seems to be greater in the mining operations, because it would appear that your conversion factors, even given the variation -- appear to be higher than theirs?

THE WITNESS: A. Around three is higher than, say -- I don't know ---

Q. Weill's; which was between one and two, as I recall.

A. I don't know if I'd want to generalize that far. I say that because, for example, in the Dement paper, Dement's paper -- he is an occupation hygiene engineer; he's probably one of the most competent people on this whole thing. He put a lot of effort into it, and he is at levels of around eight -- six to eight -- so I don't know that --

Q. Can't make that generalization.

A. --- there's any general pattern, really. You'll appreciate what variation, if you have access to this analysis which Dagbert did of Gibbs' information to the Beaudry Commission -- you will see the enormous variation there is in conversion rates, and it would be necessary, certainly, if you took Weill's conversion or Enterline's, which I'm not entirely familiar with -- you'd have to relate them to the same point on the scale as ours, in order to see whether they are higher or lower than ours. And I'm not sure what the answer would be.

Q. What do you mean by "the same point on the scale"?

A. The point on the scale in the sense that our 3.14 is based upon an average concentration of exposure of this group of workers, or whatever it was -- and, I'm sorry, I don't know; let us say it was twenty million particles -- now, at twenty million particles, Dagbert's data shows 3.14, or whatever. You then have to look at Weill's information and find out what is the average dust concentration that his workers were exposed to ---

Q. At twenty million particles.

A. Well, maybe they weren't, you see; maybe they were all at ten million particles; and, therefore, even using our data, he would have a different conversion factor. And, therefore, I don't know whether his conversion deviates from ours or not.

Q. I see.

A. I don't know at all.

The conversion is tremendously difficult, though; that's all I can say.

Q. I understand. Thank you, Dr. McDonald; you've been helpful on it -- extremely helpful on it.

Can I turn to a different topic in ---

DR. DUPRE: Before you do, could I just ask a question for clarification, in the same area?

I take it, Dr. McDonald, that, from what you've been telling us, the main hypothesis to which you subscribe, in terms of explaining the difference between, let's say, the textile part of the industry and the mining and milling part of the industry is that the fibre tends to get more divided the farther it gets away from the mine?

Have you ever heard the hypothesis that co-carinogens play a role, an increasingly important role, as you get away from the mine; have you ever heard this hypothesis

DR. DUPRE: (cont'd) discussed?

THE WITNESS: Oh, yes; I might even have written it. You know, I mean -- I think this is one of the things that people will speculate on. I don't think one could say more than speculate.

After all, what we do know is that there is a very important interaction -- if we consider lung cancer -- that there is a very important interaction between asbestos exposure and smoking.

It would not, then, be all that unlikely if there were other factors which there could be an interaction with asbestos; and I suppose it's broadly likely that the further you get away from what is a sort of rural occupation, of mining and milling, to an urban, city-dweller's occupation of working in a factory, or, in particular, applying insulation materials in the holds of ships, or around engines or in heating plants, or whatever, that certainly the opportunities for exposure to all sorts of other things exist, and probably exist in a more complex way than would be present in mining and milling; I'm again speculating, but it doesn't seem unreasonable.

And, therefore, I think you must leave open the possibility that part of the difference -- and we haven't quite come to establishing this difference -- but part of the difference which probably exists of the much higher risk of lung cancer, apparently, in some processing industries or applying industries than in mining and milling might, on the one hand, be due to other substances, or -- and, in addition -- to changes in the physical nature of the fibre; and perhaps both.

But having said that, I don't know of any evidence to confirm that.

DR. DUPRE: Now, just taking it as what it is, a hypothesis upon which to speculate, if I take that hypothesis

DR. DUPRE: (cont'd.) and speculate upon it and then ask myself how best to express a standard, were there to be a standard, if I entertained the co-carcinogen hypothesis, this would lead me, as a layman, to speculate that a standard, say, in textile or cement, might be better expressed in terms of particles than in terms of fibres.

THE WITNESS: I'm sorry; I don't quite follow why you think it would be better expressed in particles.

DR. DUPRE: Well, if you're measuring dust particles, as you pointed out, there's all kind of junk in the air, say, in the textile plant that is not necessarily asbestos fibres.

Now, if I take that to be correct -- and, of course, I can see, if you entertain another hypothesis, how this leads you to say, no; you're better there to measure in terms of fibres. But if I entertained the co-carcinogen hypothesis, wouldn't that resurrect the possibility of expressing it in dust particles?

THE WITNESS: I see your argument now. I think, then, that would imply that you feel that maybe the dust is a better measure of these co-carcinogens; but I suppose that my next speculation would be that I wouldn't think it was the dust that was the co-carcinogen. I would expect the co-carcinogen much more likely to be gases or trace metal or something of that kind, which would be just as -- you know, which the dust wouldn't be any better measure of at all -- perhaps some oils, perhaps -- whatever ...

I wouldn't, frankly -- I mean, I have to recognize this speculation of the co-carcinogen, but I still feel that, quite conceivably, the whole thing could be fairly adequately explained in terms of the actual size and shape of the fibres, conceivably. I wouldn't want to abandon that thought.

DR. DUPRE: That is the alternative hypothesis that you believe to be more plausible?

THE WITNESS: I would put it this way: that, until that one had been measured and disposed of, I wouldn't want to get too far on the other.

DR. DUPRE: Thank you, counsel.

MR. LASKIN: Thank you, Mr. Chairman.

Can we turn to, apparently, another co-carcinogen, and that is tobacco, and talk a little bit about smoking. If we go, first of all, to one of your conclusions which you expressed in your epidemiological review article, which is tab 22, at page 590, and you there show the table that Hammond et al. developed on the insulation workers' study and pit against that your own work in Quebec.

Q. Do I take it that one of the conclusions from your own work is that it doesn't lend the same strong support to multiplicative interaction, or a multiplicative model, as does Hammond's study?

THE WITNESS: A. No; I think that would be putting it too strongly. The Hammond study produces data which are -- incredibly closely agree with a multiplicative model. Our data are also compatible with a multiplicative model; indeed, our statistical colleagues now, who've been working over this again and again, now say that it is probably, on balance, more compatible with a multiplicative model than an additive one, but the ability to discriminate the two is, you know, limited; and, of course, also, there isn't -- we're just taking two very simple models here; there are all sorts of permutations and combinations of those models that one could bring up.

It's also fair to mention that, since this data's been presented, Selikoff and Hammond have produced an analysis of smoking and exposure in their amosite factory, and they find now

A. (cont'd.) data that are much more like ours. So, at the end of the day, I don't think one can be very dogmatic about this; I think one can say that there is a very important interaction between smoking and asbestos, and it could be anything between additive and multiplicative and ---

Q. Fair enough.

A. But what it does exclude -- and I think we say so here -- the important thing is, it -- both studies, all studies now are really excluding the possibility that smoking is a prerequisite for asbestos-related cancers; that you apparently can get a dose-related risk of lung cancer in people who, as far as one knows, are non-smokers.

Q. I see.

Can I come back to tab 18, page 20, and look at table 11, which I take it is your case control analysis for smoking; and you make the comment, at the bottom of the page, and, indeed, it seems demonstrated in the table, that it might appear from that table that the relative risk, due to asbestos dust exposure, was, in fact, higher for non-smokers than smokers.

Is that, do you believe, a real effect, or is there something in the selection of the controls? Why do we have that result? It seems different than most other results.

A. Now, I don't -- it's a matter of relative risk; I mean -- can I turn you back; I think it helps -- the numbers are a little simpler on that tab ---

Q. Table 9?

A. Page 590 of tab 22, from the review article. So that you go from, shall we say, one -- rather, if non-smokers with little asbestos exposure are taken as the base line of one, about seven times as much -- the risk is about seven times as great in heavy -- heavily exposed asbestos workers.

A. (cont'd.) On the other hand, in heavy smokers, of course, it goes up to twenty-five times as great.

Q. Who both smoke and are exposed to asbestos?

A. Yes; that's right.

Now, this sort of model sort of would imply that both asbestos and smoking are carcinogenic, and that there are certain susceptibles to carcinogenesis and that, up to a point, you can't -- if you expose them heavily to both, you'll begin to run into a ceiling of how many people are susceptible, so that you can't get as high -- I mean, the Hammond model tends to suggest, yes, you can; you can get just as many. You can get a full multiplicative model.

But I think the implications of ours are that you're running against a ceiling of susceptibility.

I may say, we found the same sort of thing in relation to bronchitic symptoms in asbestos workers and smoking, where heavy-smoking workers, who weren't much exposed to asbestos -- about half of them eventually developed chronic bronchitic symptoms.

Non-smoking workers, who were heavily exposed to dust, about half of them developed bronchitic symptoms. But if they were exposed to both, you still only got about half of them. In other words, it looks as if you are running up against that about half the population are susceptible to getting bronchitis, and if you hit them with two things, if you like, you overkill.

Now, I don't know if that makes any sense or not, but I would tend to look at it that way. But all this is explaining something which maybe isn't true, anyway. I mean, I do repeat that I think it's pressing this data too far to say we can apply -- interpret these figures precisely.

You see, one of the things that prevents you doing

A. (cont'd.) that is that lung cancer is very, very infrequent in non-smoking, lightly exposed asbestos workers; so you have a very, very small base on which to calculate your relative risk -- a very unstable calculation.

You'd only have to have two cases instead of one, shall we say, there and the whole thing would change. So, you know, I repeat: I think that what all the data tends to suggest is that the relative risk of asbestos exposure for non-smokers is certainly as great, if not greater, than for smokers. The relative risk; not the absolute risk. I have to emphasize that very strongly.

Q. I understand that. The absolute risk, of course, is higher ---

A. Much higher, when you get into the smoking asbestos worker. Obviously, that has medicolegal implications, too.

Q. Can I take you to page 21, tab 18, and I wanted to ask you about one of your conclusions; and in the second full paragraph on the second column of page 21, you begin to talk about "uncertainty remains over the form of interaction," and you carry on; and then the sentence about halfway down that paragraph:

> "Either model [I take it, either additive or multiplicative] allows one to speculate that the dust concentrations in the Quebec mills in the early 1950s, before effective dust suppression was introduced, carried a lung cancer risk equivalent to heavy smoking whereas, at more recent concentrations of around [one million particles per cubic foot], the order of risk may now approximate to less than one cigarette a day."

Can you amplify on that a little bit?

A. Well, it's a direct application of the equations of risk -- relating risk of lung cancer to smoking, on the one hand, or to exposure, on the other.

And I seem to think that -- I'm not sure if this was dealt with in paper 18 -- no; unfortunately, it wasn't.

Q. Perhaps the most direct way to ask it: what is the order of magnitude of the risk that you're referring to?

A. Well, what it boils down to, as a matter of fact, and why I was looking for it, because when I was presenting that paper, I did, in fact, show a graph. I put on the same graph the relative risk of lung cancer in relation to smoking, on the one hand, and in relation to asbestos, on the other, in mines and mills.

And, on that, by putting the two scales side by side, you could see that, within this industry, roughly speaking, what was the equivalent risk of so many cigarettes to so many particles and fibres, and it is by reading that off that you get this sort of thing.

I mean, what it does show is that the, again, even on our very rough data, we get a perfectly good linear response of lung cancer to smoking in the asbestos industry. We ignore the amount of asbestos exposure and just plot the risk of lung cancer against smoking, and we get a perfectly linear relationship.

If we ignore smoking and plot asbestos exposure, we get another linear relationship. So, by adjusting the scales, you can get the lines to coincide, and, therefore, you can read off, if you like, the sort of equivalents of a certain amount of exposure to a certain amount of cigarettes.

MR. LASKIN: Mr. Warren tells me that the tables that you were referring to are in tab 17.

MR. WARREN: I think it's tab 17, pages 5 and 8.

THE WITNESS: Yes; that's right. That's the one I started looking for.

MR. LASKIN: Q. It would then be, do I take it, comparing figure 2 at page 5 as against figure 4 at page 8?

THE WITNESS: A. Figure 2 -- I'm sorry; I'm now lost -- oh, I see; yes. Well, there you have -- what we've done in this figure 4 on page 8 is to adjust the scales so that this straight line satisfied both; we were able to put on it both the points for risk relating to smoking habits and the risk relating to asbestos.

Q. I see.

A. So we can, therefore -- the limitation of all this is, in this work population -- in this work population -- we can say, therefore, that the risk associated with -- let us take fifty million particles for fifty years; that is equivalent to one million particles for a lifetime -- for a working lifetime; and that, then, becomes equivalent to -- that would then be equivalent to twenty cigarettes a day. Now, I think I'm wrong here; I need to think about this. I'm sorry.

MR. LASKIN: Well, since it's one o'clock, perhaps we'll give you the opportunity over the lunch-hour.

DR. DUPRE: Dr. McDonald, is 2:15 a convenient time?

Fine; we shall reconvene at 2:15.

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DR. JOHN CORBETT McDONALD, PREVIOUSLY SWORN, RESUMES THE STAND

MR. LASKIN: I think we are waiting for Mr. Warren.

DR. DUPRE: I think maybe fifteen minutes grace is a sufficient period. Shall we proceed, please?

MR. LASKIN: Thank you, Mr. Chairman.

MR. LASKIN: Q. Dr. McDonald, can we come back to figure four, page eight, tab seventeen, which we were discussing just before lunch, and we were discussing it in relation to the conclusion that you expressed in the paper at tab eighteen, about comparative risks at today's exposure levels. Can you help us with this figure four?

THE WITNESS: A. I didn't immediately recognize what the lowest scale was. It is, what is shown there in millions of particles per cubic foot for fifty years, is the relative risk associated with a concentration at that level for fifty years, and therefore the statement in...is it...

Q. Twenty-one, I believe.

A. Twenty-one, which was to the effect that... I can't find the phrase...

Q. It is towards the end....

A. Oh, yes.

Q. ...of the last full paragraph on page twenty-one.

A. Yes. Yes. Either model allows one to speculate that the dust concentrations in Quebec mills in the early 1950's, before effective dust suppression was introduced, carried a lung cancer risk equivalent to heavy smoking.

Okay, well that means that if we say that the levels in the 1950's were around about seventy million particles, that was equivalent, you can see on this graph, to approximately thirty-two, three, cigarettes a day.

A. (cont'd.) The other part of the thing is, whereas at more recent concentrations of around one million particles, and we now have difficulty in really looking at one, but one will clearly be equivalent to about half a cigarette a day...or shall we say three or four a week. This sort of thing is not to be taken too precisely, but it gives one a sense of the rough equivalents of the hazard relating to types of exposure.

Q. Did your research demonstrate any relationship between smoking, asbestos and any other asbestos-related disease, and in particular I am thinking either of asbestosis or mesothelioma?

A. In our studies of asbestosis we have never been able to demonstrate any association either way, in smoking. We have never been able to find any evidence that smokers did worse or better than nonsmokers in relation to x-ray changes or mortality, as far as I can recall. Certainly not x-ray.

Functional changes, allowing for the cigarette smoking level, I mean there is no doubt that smokers have a less good respiratory function anyway.

In lung cancer we have reviewed that. In mesothelioma, the information on that comes from our various case control studies of mesothelioma in Canada and...in Canada, really, rather than the United States...in which we found no evidence that mesothelioma cases were related to smoking at all, and I think others who have looked at this question agree with that. I think it is generally agreed that smoking does not seem to contribute to lung cancer.

Q. To mesothelioma?

A. Mesothelioma, sorry.

Q. I note in the epidemiological review article which you wrote, you refer to the work of Hammond on assessing the effects of cessation of smoking.

A. Yes.

Q. I am wondering whether you or your colleagues have done any similar work with respect to the Quebec cohort?

A. We have never analyzed that.

Q. I wonder, Dr. McDonald, whether you are familiar with a paper that was written by Mr. Nicholson and some of his colleagues, that looked at a smaller cohort of the Quebec miners, the same cohort that you are looking at? Do you have any familiarity with that particular work?

A. Not close familiarity. I have looked at the data and it was presented at the same meeting as this paper here on...

Q. Paper seventeen?

A. Paper seventeen.

Q. I wonder if I might show you a copy of this article which is entitled, Long Term Mortality Experience of Chrysotile Miners and Millers in Thetford Mines, Quebec.

A. Well...

DR. DUPRE: Is this, counsel, a paper that will be in the exhibits that will accompany Dr. Nicholson next week?

MR. LASKIN: It is, Mr. Chairman. I have some extra copies here for counsel.

THE WITNESS: A. I wouldn't like to comment on it in any detail at this point, and I would need to study it further, but it is my recollection that the findings in this study were essentially the same as our own, in mortality. I think they were entirely compatible with the mortality experience of the entire cohort.

Of course, his selection of the cohort, of the subcohort, was very different from ours.

MR. LASKIN: Q. As I understand it, he took all men who were employed during the year 1961, who had at least twenty years seniority?

A. That's right.

Q. Then assessed them as of 1977, which I take it then would give a latency period of at least thirty-six years from first exposure? Is that your understanding?

A. Yes. It isn't quite that simple because it was only in Thetford Mines, and it was only in...I think it was the members of one particular trade union, if I recall.

Q. Yes.

A. It indeed involved, I think, only two or three of the companies, so it isn't so easy for us to establish exact comparability. But bear in mind that Thetford Mines is where all our excess mortality did occur, virtually, and in long-term people, and the standardized mortality rates for lung cancer which he observed were quite compatible with our own.

I think the other point to note is that he also found a very marked deficiency of mesotheliomas.

Q. Yes.

A. I think I have discussed this with Dr. Selikoff on occasion and I don't think there is any important difference in these findings from our own.

Q. There are only one or two things I really wanted to talk about in relation to the paper, and I appreciate...I will not get into any detail with it.

I take it that yes, your findings were roughly similar if you took a cohort that had been similarly exposed for a long period of time.

A. Certainly.

Q. I am going to come back to mesothelioma in a little more detail in a few moments, but can I take you to page nineteen of this paper for just a moment?

It would appear that the authors are there trying to deal with the low incidence of mesothelioma in Quebec, as opposed to their rather higher incidence in their own insulation

Q. (cont'd.) worker study, and I take it trying to deal with the question of whether it is solely attributable to fiber type. Can I take you just to the last paragraph on page 19, where the authors expressed the view that, "an alternative source of the differences may lie in the differences in fiber size distributions in the three work environments," and carry on from there.

Does that have some of the flavor of what you were talking about this morning, about how the fibers do change as they go through different processes farther away from the mill?

A. Yes. I think this is quite right, that anytime we are looking at the effects of asbestos exposure we have got to bear in mind fiber type, and the process, and the process because of the fact that it means that the asbestos product is likely to be physically different in different processes.

So I would, without reading this, I would guess that this is bringing up what would be, to my mind, a perfectly reasonable possibility that perhaps the difference could be due to the difference in form of the fiber.

I think that has been the state of affairs, really, until quite recently where...because there has been a marked deficiency of information on mesotheliomas in manufacturing industries. I think that the thing that puts the nail in this coffin is the fact that in the South Carolina plant, which has probably the highest risk of lung cancer yet observed, there is still virtually no mesothelioma. There is one case.

So I think that removes that possibility, in my opinion.

Q. In other words, what you are saying is that the nail in the coffin, you mean the alternative source explanation...

A. That it is purely a matter of the fiber being

A. (cont'd.) different, if you like, that the insulation workers had more mesotheliomas because the fibers were different, the chrysotile fibers were different in insulation work, and that might account for their high mesothelioma risk.

My point would be that if that is the case, then why didn't the textile workers of South Carolina have a very high mesothelioma risk.

Q. Do I take it then the conclusion is, and we may develop this in more detail in a few moments, but the conclusion is that fiber type has a very definite effect here?

A. I think that's inescapable, frankly.

Q. I take it...the Dement study has already been marked as exhibit number four in these proceedings, and I take it you refer to the conclusion which is found right near the end of the paper, to the effect that only one of one hundred and ninety-one deaths in Dement's study was attributable to mesothelioma?

A. Right.

I can say that a much larger study than Dement's has been done in the same plant, with the same result.

Q. With the same result?

A. Mmm-hmm. Still the same one mesothelioma, the same one.

Q. Still the same one mesothelioma?

A. Yes.

Q. I gather that is being done independently by your wife?

A. Yes.

Q. Is it, is that study likely to be available for publication or discussion?

A. Well, a preliminary report has been made public. This is a study which has...she has studied three American plants, one using chrysotile only, which is this one; another one using pretty well chrysotile only, but in a friction plant; and another

A. (cont'd.) one using chrysotile, amosite and crocidolite. The two chrysotile plants have only got one mesothelioma between them in a very large series of deaths, whereas the...I have forgotten the exact numbers, but it's eighteen or twenty mesotheliomas in about half the number of deaths in the crocidolite mix.

Q. We'll certainly look forward to hearing about that in late August.

Do I take it in a general way that Dr. Allison McDonald's work in the South Carolina plant produces the same kind of general conclusions as the Dement paper in exhibit four?

A. Yes, with all its problems.

Q. With all its problems.

A. Yes.

Q. The one other matter I just wanted to touch on in relation to the Nicholson paper, is at page 15 of the paper, and in particular table four.

As I understand it, Nicholson attempted to correct death certificates by going behind the death certificate?

A. Mmm-hmm.

Q. Which is a method you didn't employ, and I take it one of the reasons is that that affects the comparison that you've got?

A. No, we did employ it, too, but we didn't use the data because it would not allow...you would not be permitted to...if you correct your death certificates in the light of other information, you cannot use the figures from national or provincial death rates, because they are not based on correct death certificates.

Q. You get an invalid comparison?

A. Well, we did do the same thing to see what would be the effect.

Q. Did you..the one thing that struck me about

Q. (cont'd.) table four and Nicholson's work, was that in his cohort, which was only some five hundred and forty-four people, he found a hundred and thirty deaths. The correction for asbestosis seemed to me to be fairly significant, from seventeen upwards to twenty-four.

A. I wasn't aware of that...

Q. As I read his paper I am just wondering, did you find any of a similar kind of experience when you corrected death certificates?

A. We made no attempt to correct deaths from asbestosis. We made no attempt even to look at them. We were only concerned with looking at to what extent lung cancers were incorrectly certified, so we did look to see whether autopsy data would have added any significant number of lung cancers, or indeed removed some. There was a small change in the direction you would expect. There were more cases found at autopsy.

Q. But not a...

A. Not many. About half a dozen, or something. I think it's in the paper.

No, but I don't know, I wasn't aware that they had looked at cause of death due to, ascribed to asbestosis and corrected that. I would think that would be quite a difficult operation, and I don't know how they did that.

DR. DUPRE: May I ask a question, counsel?

MR. LASKIN: Sure.

DR. DUPRE: Do I take it, Dr. McDonald, that you were just saying that you did not look at any of the asbestosis deaths, for example, in terms of the way they are reported in tab eighteen? For example, if we look at tab eighteen on page 19...

THE WITNESS: Is this in the...page 19, yes. Pneumoconiosis, twenty-five, twelve, twenty-seven cases?

DR. DUPRE: Yes, I'm looking at table eight.

THE WITNESS: Table eight, yes.

DR. DUPRE: Can I ask this? You list, for example, under Cause of Death in table eight, heart disease?

THE WITNESS: Yes.

DR. DUPRE: You also list pneumoconiosis, which obviously would include all asbestosis cases?

THE WITNESS: Yes.

DR. DUPRE: Now, in terms of the way that deaths are reported here, if an asbestotic died of a heart condition that was induced by the asbestosis, do you know here if that death would have been recorded under pneumoconiosis, or would it have been recorded under heart disease? Or would you just take whatever was on the death certificate?

THE WITNESS: We don't take what's on the death certificate. What you take is what the coder coded what was on the death certificate. I refer to table two, which perhaps... page 50...which does give the distribution of our deaths, of all four thousand-something deaths, by four thousand, four hundred and sixty-three deaths, by certified cause and the International Classification of Disease Code, and we don't put any interpretation on that. In other words, what we get is the classifier, that is, the Quebec...for the most part we used the Quebec government classifying person...to code these...and in fact we used the code as they were used in the Quebec statistics.

There are rules, of course, I think, which the coder should apply correctly...doesn't always, but should apply correctly. If I recall, for example, I think pneumoconiosis, which if it is mentioned as one of the causes of death, will take priority. I think even if you have had heart disease due to pneumoconiosis, then pneumoconiosis would become the primary cause of death. Just as cancer tends to take priority over almost everything if it is mentioned. But there are rules, and...

DR. UFFEN: Does asbestosis have a code number?

THE WITNESS: I think, as a matter of fact, you'll see here codes of five two three and five two four...I can't remember offhand...I've got a feeling that asbestosis only comes into the next series of digits. I'm not sure about this.

DR. UFFEN: It wouldn't take priority over pneumoconiosis, then?

THE WITNESS: No. No, it would be a form of pneumoconiosis...I think...some people in this room would probably know the answer straight off, but I think that asbestosis will be five two three point five, or something, as opposed to five two three one, which may be silicosis or whatever.

I'm not sure of this, but I think that's the way it works. But no, it wouldn't. It would be a subdivision of pneumoconiosis.

What I don't know is the difference, offhand, between five two three and five two four. I've got a feeling that five two three...one may be specified and the other may be unspecified, but I don't know.

DR. UFFEN: In other words, if they are examining patients where they didn't really expect to find asbestosis, could many of them be classified as pneumoconiosis? There having been no post mortem or anything else, they would not be identified as asbestosis?

THE WITNESS: I think it's sort of unlikely that that's what would happen. I think that if a case has an illness which is compatible with pneumoconiosis, the certifying physician will either put down pneumoconiosis general, or if he has taken the history and found it was an asbestos worker, he may jump to conclusions it is asbestosis.

DR. UFFEN: Suppose he wasn't an asbestos worker?

THE WITNESS: This is a type of, I think that in practice the term pneumoconiosis is a very dubious diagnosis,

THE WITNESS: (cont'd.) because it gets diagnosed in relation to what the physician thinks is reasonable. If the man...if he doesn't know that he has ever worked with dust, he probably won't suspect it.

This could be one of the reasons why we find a very nice linear relationship for pneumoconiosis, because it may reflect directly the physician's conviction that the man was an asbestos worker, and the more certain he is that he is one, then he would be more likely to put that.

I don't know that for certain.

The rest goes with lung cancer. There is another sort of bias there.

But I mean, the quick answer is, we used the certified cause and the cause as classified, but this...if I recall, almost all forty-six cases were, the word asbestosis was mentioned on the certificate. Not in all of them, there were a few where it wasn't.

But if in, let us say heart disease, the word... supposing the person was said to have died from coronary heart disease due to pneumoconiosis, I believe the way that would work is that that would be classified as pneumoconiosis.

If on the other hand the classification...the certificate said, cause of death - pneumoconiosis, heart disease due to...no, heart disease...let's put something else...pneumonia due to heart disease, and on the third line of the death certificate - associated conditions, because there is an opportunity to put associated conditions, they don't take priority, so there would be death certificates in which asbestosis is mentioned as an associated condition, which does not get coded to them.

For example, in several of the deaths due to other respiratory causes, the word asbestosis might be mentioned on the associated conditions. But if a physician did not attribute

THE WITNESS: (cont'd.) it in part of the changed causation, it wouldn't be so coded.

DR. UFFEN: Would you like to move to tab fifteen?

MR. LASKIN: Well...

DR. UFFEN: There are quite a few changes..

MR. LASKIN: I will. I just wanted to ask just one more question on that.

MR. LASKIN: Q. I noted at tab eighteen, page 22, you raised the possibility that there may have been an over-diagnosis of asbestosis amongst your more heavily exposed in your cohort. I suppose what I'm trying to do is reconcile that with what appears to be the findings of Nicholson, et al, on what seems to be an underdiagnosis.

THE WITNESS: A. I can't comment. I mean, I have no idea what mechanism they used for saying this case ought to be attributed to pneumoconiosis rather than what was on the death certificate.

I suppose one way one might consider it is if you had somebody that the death certificate said heart disease, but on the death certificate, associated condition, pneumoconiosis, that perhaps Nicholson decided it shouldn't have said heart disease, it should have said pneumoconiosis.

Q. Yes.

A. It's a matter of opinion.

Q. I think it would become a judgement matter.

A. I don't know, I have really no idea.

Q. No, and it doesn't appear from the paper as to how they did it or what they did.

A. I wouldn't want you to think that we...that I'm contradicting what I've written...but I wouldn't want you to think that we would feel that maybe asbestosis...I mean, I think that this sort of bias would go so that there would be a tendency for persons to be diagnosed as pneumoconiosis if they had a clear

A. (cont'd.) history of heavy dust exposure.

On the other hand, it is possible that there might be an underreporting of pneumoconiosis in places where they didn't have that history. So I don't know what the net result...

MR. LASKIN: Mr. Chairman, I wonder if before I move on, just to keep the record clear, I would mark this paper that we have referred to, by Nicholson, et al, as...I think the next exhibit is exhibit nineteen. We may have gotten out of order at the beginning.

Miss Kahn tells me that the first two exhibits this morning should have been seventeen and eighteen, rather than sixteen and seventeen.

DR. DUPRE: Does anybody here understand this?

MISS KAHN: Just to review what we've done so far today then, exhibit seventeen is Dr. McDonald's curriculum vitae, exhibit eighteen is Dr. McDonald's compendium of articles, this article by William Nicholson now becomes exhibit nineteen.

MR. WARREN: I would like to make a suggestion, just for simplicity, since this is a reference to Dr. Nicholson's testimony and since it will be put in, why don't you just refer to it...why don't you put in all of Nicholson's now as nineteen, and then refer to this as nineteen A...just so we won't have it twice in the record, so we won't be citing to it in a duplicate fashion.

DR. DUPRE: The problem is that the Nicholson book is not yet ready.

MR. LASKIN: It is not ready, but I think to simplify it, it is item number eight on Dr. Nicholson's index, and let's refer it that way, and it will...

DR. DUPRE: It will be tab eight when we get the Nicholson exhibit.

MR. CASGRAIN: It will be tab eight then?

DR. DUPRE: It will be tab eight in the Nicholson book.

MR. WARREN: Could we take just one additional step to make it clear when we refer to it in the record, to put Nicholson...give Nicholson number nineteen right now, so that this becomes tab 8, so that when this study gets referred to from here on out, it's clear what it is.

MR. LASKIN: Fair enough.

MR. CASGRAIN: I think it is also important to make sure that this study is not annexed to Dr. McDonald's own study, and it seems prior to that as if the two were corresponding, which is simply not the case.

MR. LASKIN: Not at all.

MR. CASGRAIN: On which I may have some questions myself from Dr. Nicholson, by the way.

DR. DUPRE: So what we were discussing then, when we were discussing the Nicholson paper, was exhibit nineteen, tab eight.

MR. LASKIN: Correct.

DR. DUPRE: Thank you.

MR. LASKIN: Thank you, Mr. Chairman.

EXHIBIT # 19, Tab 8: The abovementioned document was then produced and marked.

MR. LASKIN: Q. Dr. McDonald, I believe the comment that the Commissioner, Dr. Uffen, raised, wished to pursue, related to your article at tab fifteen, which is entitled, Mortality After Long Exposure to Cummingtonite Grunerite.

I don't know whether I've read this article correctly, perhaps you can help me. Is it behind this article that there may be other mining operations, mining operations other than pure asbestos mining operations, such as iron or gold, that when mined will produce fibers that are asbestos-type fibers or have the same inorganic properties and may have the same kinds

Q. (cont'd.) of health effects. I've put that rather crudely, but I think you may understand the thrust of what I'm trying to get at.

The reason I ask it, and I'm sorry to make this so long, but the reason I ask it is, of course, Ontario has at the present time no pure asbestos mining, but Ontario certainly has a fair bit of gold mining and a fair bit of other kinds of mining, and I suppose my question is, is there a problem in those mining operations?

THE WITNESS: A. What lay behind this particular study was the issue, really, of what is asbestos. It arose over the question of the mining of iron ore in Minnesota, which for the last twenty-five years or so now produces a very significant proportion of all North American iron. I don't know what proportion it is, but it's a very big proportion, and the particular mineral which is used for the extraction of iron there has quite close relationships to amosite. I really hesitate to discuss this in the presence of a man who knows about minerals, but the whole trouble is that asbestos really mineralogically doesn't mean very much. It is really any fibrous mineral, fibrous silicate I think probably, fibrous mineral, fibrous silicate, which is commercially used. There is the element of commercial use that comes into the definition of asbestos.

So the iron ore of Minnesota is certainly not used for its fibrous quality. Indeed, the question does arise, what is a fiber. It is often sort of conveniently glossed over by saying that anything that is three times longer than it is broad is a fiber.

Well, I'm not knowledgeable enough to enter into that discussion, but certainly there is an awful lot of difference between the chips of the mineral iron which get produced, and asbestos as used commercially for its fibrous qualities.

Nevertheless, for example, in the deposits in

A. (cont'd.) Minnesota, there are pieces where there is truly fibrous material which I think is probably indistinguishable from what the South Africans call amosite.

Now, there was a lot of concern, and you may recall, in this area, which arose mainly because of the contamination of Lake...whatever it was.

Q. Lake Superior.

A. Lake Superior, by the tailings from the big iron mining in that area, and a lot of concern that this material might indeed carry the hazards of asbestos.

Q. Was one of the things you were trying to do in this paper to find out whether that was the case?

A. Yes, in part. In part. The thing was that we were interested, on the face of it, in the type of mineral that was being pushed into Lake Superior...and I'm not trying to defend putting it into Lake Superior at all, or into the air around those parts...it was simply that it did seem inherently unlikely that this was very similar to the kind of asbestos fiber that was causing cancer and so on.

Now, the trouble was, there wasn't any easy way of investigating that in Minnesota, because they had only been operating for twenty years and therefore we didn't have a population that we could look at to see. It was too soon to see.

There had been...the National Cancer Institute in the U.S.A. had been doing some work on this and looking for evidence of any excess cancer mortality in those parts. I think so far, negatively.

But we did...it was brought to our attention that there was a mine, quite a famous gold mine in South Dakota...The Home State Mine, a most romantic place...which had been working for a hundred years and where they in fact looked for the gold through an ore, this cummingtonite-grunerite stuff, which is extremely similar, as I understand it, to the iron ore of

A. (cont'd.) Minnesota. Again, the same sort of problem, so that in this gold mine there are certainly chips and pieces that are longer than they are broad and that might meet some sort of definition of fiber. There are even small areas where there is truly chrystalline, fibrous material such as might well be classified as amosite. But it's very small amounts.

Okay. So at least we had the opportunity to take a working group there, that had had this very long exposure and really had quite good records, to see whether there was any evidence of...well, to see what their mortality experience was. So that's what it boils down to.

Of course, if this kind of material carried the sort of hazards of fibrous asbestos, the health implications, industrial implications of this would make asbestos look like nothing. This is an enormously important...so I'm told...something of the order of half the iron of North America is produced that way.

Well, sufficient to say we did look and we didn't find any important excess of lung cancer, and we found one possible mesothelioma. There it is, that's where the thing stands.

I may say that there was a bit of controversy about all this because the National Institute of Occupational Health and Safety in the U.S. had done a small study which gave a rather strong positive answer. There was some, in my view, inconsistencies, and there have since been contracted to somewhere in California, a much larger study of this population. I've seen the preliminary results of that, which I believe are exactly the same as ours.

However, the industrial implications of this are enormous, as to what is asbestos.

Q. I see you did find some kind of dust exposure relationship for pneumoconiosis?

A. Oh, enormously so. You see, these men were

A. (cont'd.) hard rock miners. There is a very significant amount of silica in the air, and they have a very large excess mortality from pneumoconiosis and tuberculosis. They have the classical picture of a severe past hazard from silicosis and silicotuberculosis.

Q. I see.

DR. UFFEN: This gives rise to the concern about whether pneumoconiosis would include asbestos cases, but not identified so on the death certificates.

THE WITNESS: Yes.

DR. UFFEN: In other words, people would think they died of silicosis?

THE WITNESS: Yes, that's right. This is quite right, and of course we get exactly the same kind of condition in reverse if a man dies in the Eastern Townships of Quebec. He'll be called asbestosis. If he dies near this mine in South Dakota, he'll be called silicosis.

But that's one other reason, if you like, for classifying them as pneumoconiosis and not worrying about the finer point.

But, I may say that we do discuss somewhere here that the classical picture of silicosis is different from asbestosis.

DR. UFFEN: The symptoms?

THE WITNESS: Yeah. The symptoms, perhaps less so, but the actual distribution of fibrosis in the lungs is a different sort of picture from asbestosis. I'm not saying there couldn't be some overlap, but the characteristic picture of silicosis is a much more intense, localized areas of intense fibrosis. You don't tend to get that in asbestosis. You get a more diffuse pattern, particularly over the bases of the lungs.

The other point is, that...and it's brought out in this grunerite study...that there is well-known the very

A. (cont'd.) relationship between silicosis and tuberculosis, so that as you see here, the excess mortality is about half of it due to, ascribed to silicosis, and I should think a lot of them were also silicotuberculosis, really, and quite a lot to tuberculosis, probably with associated condition silicosis. And you have this interaction of silica and tuberculosis which doesn't seem to be present with asbestos, or if it is, not very dramatically.

DR. UFFEN: On the first page, 271 of tab fifteen, in the introduction, righthand side below the summary, there is a statement that says, "These resemble amosite fibers in appearance and chemical constitution, although lacking their strength and elasticity. They are mostly less than five microns in length".

THE WITNESS: Yes.

DR. UFFEN: Is that significant?

THE WITNESS: Well, yes. I think the fact that they are short, on the one hand, will probably increase probabilities of inhalation. On the other hand, the fact that they are short will perhaps be likely to reduce their biological effect. Such evidence as there is tends to suggest that it is longer fibers that have a bigger effect.

DR. UFFEN: If I remember all the things we are being taught, is it five microns that's the limit at which the measurements are made for regulatory purposes?

THE WITNESS: I think commonly, yes.

DR. UFFEN: That's why you would use five?

THE WITNESS: Yes.

DR. UFFEN: May I proceed? Just a couple more.

The next page, 272, again righthand side at the top, it's discussing the amount of such dust, and it says,

"These amphibole minerals which are chemically and chrystallographically similar to commercial

DR. UFFEN: (cont'd.) "amosite, have been shown by Gillam and coworkers, and Dement and associates, to be present in the mine. They reported that by analytical transmission microscopy, eighty to ninety percent of airborne fibers in the mine demonstrated an amphibole diffraction pattern...", etc.

That seems to be quite large, doesn't it? Eighty to ninety percent of the dust was essentially amosite?

THE WITNESS: I'm not an expert on this, but I think this would imply that they are chemically similar to amosite, rather than physically. I don't think x-ray diffraction would tell you anything about their structure. I mean, you could in fact...you would do that by some sort of visualization.

DR. UFFEN: But do we understand...have I got it wrong..the very first paragraph, the introduction, it said that,

"The process of crushing up the rock, the cummingtonite grunerite is turned into the fibrous form by virtue of its cleavage."

Then we end up with this dust which is ninety percent equivalent to amosite. That implies to me that it's both physical, as well as chemical.

THE WITNESS: Yes, I think that that would be probably not true.

DR. UFFEN: But I interpret this correctly that this was the basis of the disagreement between your study and the study of Gillam and Dement? Or were there other kinds of disagreement?

THE WITNESS: Oh, no. The only disagreement was in the finding. This...what you just in fact quoted to me is a quote from them. We didn't do any studies of this kind. This is what Gillam and others reported. They reported that by analytical transmission microscopy...I mean, their reason for being concerned was that they said this is chemically similar

THE WITNESS: (cont'd.) to amosite, and indeed I think mineralogically the series has much in common, but what I think it all boils down to is that chemically it may be similar, but physically it's very different. I mean, most of the stuff was in fact the little cleavage fragments, little chips, you know, that don't look at all like a fiber.

Now, having said all that, within the deposits of cummingtonite grunerite and so on, there are areas, small pockets, in which there is true fibrous amosite. But that would be a very small proportion of the total. It is just analogous to saying that in chrysotile mines that you electron diffraction on the dust, the serpentine dust, and say that's the same as the chrysotile. Well, it is chemically identical, but it's, of course, physically completely different.

DR. UFFEN: One final...is this the same Dement that has been referred to...

THE WITNESS Yes, yes.

DR. UFFEN: ...who just got his doctorate?

THE WITNESS: Yes, yes.

DR. UFFEN: Thank you.

MR. LASKIN: The author of exhibit four.

THE WITNESS: Yes.

MR. LASKIN: Q. May I turn, Dr. McDonald, to the subject of mesothelioma in a little more detail, and I take it one of the rather striking statistics from your own research was to the effect that in your very large cohort in Quebec, of over eleven thousand workers, you found only, I believe, eleven cases of mesothelioma...all of those workers being exposed only to chrysotile? Whereas in your gas mask worker study of some hundred and ninety-nine employees, all of whom were exposed to at least some crocidolite, although over a relatively short period of time, there were nine cases of mesothelioma? Have I

Q. (cont'd.) accurately summarized that?

THE WITNESS: A. Yes, you've understated it actually, because even of the cases in the Quebec group they were, some of those cases had been exposed to crocidolite.

Q. The other...another main conclusion which appears to emerge from your research is that comparatively, and we touched on this before, the incidence of mesothelioma in your cohort is very much lower than in insulators, factory workers and so on, leaving aside Dement for a moment?

A. Not leaving aside Dement. It is included, exactly the same as mine.

Q. But you and Dement stand at the low end of the spectrum, as I understand it, in terms of incidence rates and other cohorts seemed to produce a higher relative rate?

A. No chrysotile cohort has produced a high rate.

Q. No chrysotile cohort?

A. Yes.

Q. You have been kind enough to bring one additional article which perhaps we ought to have included and didn't. I wonder, because it's got such a well laid out table, if I might show this to you. It's your article entitled, Mesothelioma as an Index of Asbestos Impact.

DR. DUPRE: Is this exhibit twenty now?

MR. LASKIN: I think...may we mark this as exhibit twenty.

MR. WARREN: Excuse me, Mr. Chairman. I wonder if it wouldn't, once again just to be systematic, make sense to add this onto eighteen as tab thirty?

DR. DUPRE: All right. As you prefer.

MR. LASKIN: Twenty-nine.

MR. WARREN: Twenty-nine, I'm sorry. Twenty-nine.

DR. DUPRE: So this is not exhibit twenty. It is now officially exhibit...

MR. LASKIN: It's going to be exhibit eighteen, tab twenty-nine.

DR. DUPRE: Exhibit eighteen, tab twenty-nine.

EXHIBIT # 18, TAB 29: The abovementioned document was then produced and marked.

MR. LASKIN: I'm going to let Mr. Warren mark all my exhibits from here on in.

MR. WARREN: It's just so much easier to have a record that is consistent in its pattern.

MR. LASKIN: Q. Now, as I understand it, Dr. McDonald, this is a paper which you gave at the Banbury Conference earlier this year in Coldspring Harbour. That's correct?

THE WITNESS: A. Correct.

Q. That was the conference, I gather, which collected together a number of people to deal with the so-called United States Estimates Paper. We have already heard from Dr. Enterline, who talked about the paper he gave. Was this paper given at the same conference?

A. The same session of the same conference.

Q. The same session. Can you just tell us briefly, bearing in mind none of us will have read this paper, just what you tried to do in this paper?

A. Yes. The background, of course, is that there have been attempts to estimate the contribution of asbestos exposure to human cancer, particularly in the United States. One estimate which was, I think, presented by the Secretary of California, was very controversial in that it was...I can't remember the exact figures, but it produced a very high estimate of the contribution of occupational factors to cancer, and in

A. (cont'd.) particular focussed on a number of carcinogens, of which one was asbestos. I don't remember what estimate he produced, but shall we...let us say it's on the order of thirteen or fourteen percent, if I remember, of all cancer in the United States attributable to asbestos exposure.

Now, that was criticized by quite a number of people. So, at this meeting there were about half a dozen papers, of which ourswas one, which were attempts to estimate the contribution, in this particular session, of asbestos to cancer.

Different people took different approaches to this, and it was such a very full conference that I certainly wouldn't want to comment on the others, because I hardly remember them. I'm looking forward to reading their papers in detail, of which Dr. Enterline was one. There were, in fact, I think, half a dozen and it's worth noting that they did cover a range of opinions.

Q. Can you tell us about your approach?

A. The approach that I took...that my wife and I took...was, we have been struck with the fact that mesotheliomas are fairly clearly definable. I don't mean that they are that easy to ascertain and there aren't diagnostic problems, but they are rather dramatic tumors, universally fatal and fatal within a short time.

We have been struck with the fact that in cohort studies of asbestos workers, you can look at the excess mortality from various causes...for example, excess deaths from lung cancer, gastrointestinal cancer, whatever you look, asbestosis... and also look at the mesotheliomas. Almost by definition, the mesotheliomas are taken to be excess, because in most cohort studies you would not expect any important number.

If you look at the cohort studies and the table which I think I really would wish to mention, to look at perhaps later, is a table in which we tried to bring together and summarize

A. (cont'd.) the results of all known, of all available cohorts.

Q. I think it's at page five, in the article anyway.

A. That's right. I think these are still proofs, so they haven't probably got a page number yet.

What we did was to look at the ratio of mesotheliomas to excess mortality from respiratory cancers and digestive cancers. Although there is a fair range, in fact for quite a number of studies the range is not a very wide one. So that, for example, there is very...most excesses of lung cancer will lie in the range of about, for every mesothelioma, about one to five lung cancers. And for every mesothelioma...I've forgotten what number...but a smaller number of gastrointestinal cancers.

So what we then did was to use that information and say let us look at the best available estimates of the frequency of mesothelioma in North America, and let us apply those ratios and thereby estimate roughly how many excess respiratory and gastrointestinal cancers there probably are as well.

Q. What was your estimate?

A. Well, our estimate...we can either present it in a horrifying way or a nonhorrifying way...we can say that our estimate was that in males, our best estimate was about one percent, one point four percent was our best estimate of the proportions of these cancers, including mesothelioma, attributable to asbestos exposure. The more horrifying way of looking at it, that means about four thousand deaths a year at the present time in North America.

Of course, that has to be put against the fact that an awful lot of people die from cancer in North America. So about one point four percent.

With a range...our range went from point five percent to two point nine percent, and I would say that overall we were tending to somewhat overestimate. I mean, I don't think...

A. (cont'd.) when in doubt, we put it up, let's put it that way.

Nevertheless, I don't think it's a bad estimate.

The important issues, I think, are that the five other papers which estimated the same thing by all of them quite different methods, the range of answers was very similar.

Q. These are the other papers at the Banbury Conference?

A. The other papers, of which Dr. Enterline's was one.

One of the reasons which attracted us to this method was that the other methods nearly all are based on trying to estimate how many people are exposed to asbestos, and that is a very difficult thing to do. The original, what I would call erroneous estimate, was, I think, based upon taking what was a sort of maximal figure of exposed people...not an incorrect figure, I mean what proportion of American workers in the course of their work are exposed to asbestos... but of course their range of exposures was enormously variable. But what they did then was essentially to apply the experience of insulation workers to that, and that, not surprisingly, gave a very high estimate of the amount of cancer.

However, it is sufficient to say that there was, I think, fairly general agreement at this meeting, of those present, that this is around about what was agreed.

Q. Can we talk for just a moment about some of the ascertainment problems with relation to mesothelioma? As I understand it, it was not part of the International Classification of Diseases until sometime in the 1960's. Would that, in your professional opinion, have affected your diagnosis of the incidence of mesothelioma in your cohort?

A. In our cohort?

Q. Yes.

A. No. No. Because we didn't...indeed I wouldn't say that the problem of coding has any great effect on ascertainment. Not coding as such. The problem of ascertainment really depends on whether people suspect there might be a mesothelioma, and you don't suspect it if you don't believe there are such things.

So, I think that would not be the problem in our cohort, and I don't think we would have been affected in that way because we really reviewed all our causes of death as carefully as we could for any possible evidence that they were mesotheliomas, you see. Because although I have said earlier we never corrected any death certificates for the general run of deaths, for mesotheliomas we didn't apply the national rates. For mesotheliomas we sort of went through all suspected deaths and looked to see if there were any mesotheliomas, and worked on the principle that if there were any, they were in excess.

Q. How did you do that? Did you go to the autopsy reports?

A. Autopsy reports. But we also had another source, and that is, as you probably are aware, since mid-1960's, end of the 1960's, we have been trying to ascertain all mesotheliomas from pathologists in Canada, and so we had got, in addition to what you might call following people to see what they died of, we also had all the known cases of mesotheliomas notified to us from pathologists. There is some evidence that in Quebec they overestimated.

Q. I noted that, but I also noted another conclusion of yours in one of your earlier papers that on a general level you felt that pathologists were underestimating, overall, the incidence of mesothelioma, and I think the figure you used was about fifty percent?

A. Yes. That was our opinion.

Q. Do you still feel that that's true today?

A. Yes, probably.

MR. LASKIN: I think Dr. Mustard has a question.

DR. MUSTARD: Can I ask a question for clarification? What data do you have about the proportion of people dying of what was thought to be cancer, that are autopsied? Do all Canadians who die of cancer have autopsies?

THE WITNESS: By no means.

DR. MUSTARD: Is it not possible then that mesotheliomas could be missed because post mortems are not carried out on everybody who dies from cancer?

THE WITNESS Yes, definitely.

DR. MUSTARD: You have no idea of the proportion?

THE WITNESS: I think the present rate in cancer...in people who are certified cancer in Canada, I think the proportion is around about twenty-eight percent, but I'm not absolutely sure. It's higher for cancer than other causes. It may be even higher than that. I would like notice of that question.

MR. LASKIN: Q. Do I take it fairly that the major explanation that you put forward for the differences in incidence of mesothelioma is fiber type?

THE WITNESS: A. Yes. Yes. I was trying to think whether I would want to put anything in for fiber process. I don't think I would. I think it's all fiber type.

Q. In your opinion is it the fact that chrysotile is less hazardous in producing mesothelioma alone, or is there also a differential in terms of latency period involved here?

In other words, could it be that we are going to see more mesotheliomas from chrysotile in the future, and it's simply that there is a longer latency period?

A. I think it's very unlikely. I think the range of...in point of fact I think that it could be that mesotheliomas will turn out to be even more frequent in the crocidolite cohorts when they are followed up as long as the chrysotile have.

A. (cont'd.) I don't think it's that way around, frankly.

I mean, I think one has to bear in mind, for example, in the gas mask workers, they haven't been followed anything as long as the Canadian miners and millers.

Q. Are you continuing to follow that cohort?

A. No.

Q. No?

A. That proved a very difficult cohort to follow at all. There was a lot of opposition to us doing it.

Q. I take it you accept the proposition that chrysotile asbestos can cause mesothelioma and it's simply that the incidence is much smaller...it's a much rarer event...or do you take issue as to whether chrysotile asbestos can cause it at all?

A. I don't want to take issue with it. I think I would be perfectly content to say that it may well cause a small frequency of mesotheliomas. But having said that, I know of no evidence that it actually causes any.

That's taking an extreme view, but if I were to say where is the evidence that chrysotile itself causes mesothelioma, I don't know of any good evidence.

Q. In terms of the cases in your own cohort, were you able to trace most or all of them to some exposure to crocidolite?

A. Oh, no. No, no. Not at all. The majority have only a history of chrysotile exposure, so you see therefore I'm saying the majority, let us say of that cohort, and I don't remember the exact figures, say there were ten cases in males, I think in three or four there was history of a crocidolite contact. But for six or seven, there is no such history.

But we then come up against two problems, that people do not know what they are exposed to. I was told, for

A. (cont'd.) example, that in the chrysotile mills it is pure chrysotile exposure. I can remember on my first visit to one of the mills, there was a sack of crocidolite in one corner.

Now why was it there? It was there for laboratory work. So that one...you can't say that those people were never exposed to crocidolite, and my impression is that a very small amount of crocidolite can go a long way, unfortunately.

The other thing is that if you subject a population to very intensive scrutiny, which we have done, you then come up against the problem of how many mesotheliomas would you actually expect in that population, subject to the same level of scrutiny. I think the ordinary frequency seen in the general population is probably a fairly substantial underestimate.

I'm just taking the devil's advocate position here, that if you really tied me down and said can you prove that chrysotile causes mesothelioma, I couldn't. But on the other hand, in chrysotile miners who, on the fact of it, do not have any other exposure, there are cases.

I suppose the other way on is to say, I would be surprised if there weren't, because after all, even though chrysotile fiber may be by and large the wrong shape, I would expect some of it was the right shape.

Another point to remember is that even chrysotile miners are not only exposed to chrysotile, nor to asbestos. There is an important contribution of tremobile, fibrous tremolite in the Canadian mines, and fibrous tremolite is an amphibole.

So, you know, I don't know if this is an awfully fruitful argument. I think what we can really say is that there is a very low frequency of confirmed chrysotile-proved cases.

Q. I take it that one of...just on that point of exposure to matters like tremolite...I take it one of the studies you carried out involved Dr. Pooley, whom we heard from

Q. (cont'd.) Mr. Berry did some work with him in looking at fiber tissue in the lungs. I wonder if I could first of all take you to your epidemiological review article, which is tab twenty-two, at page 593, in table one.

A. The page again, please?

Q. 593, I'm sorry, Dr. McDonald.

A. Yes.

Q. Do I take it that that table at the bottom here is part of the work that Dr. Pooley did with you?

A. Yes. There is...this is the preliminary data. The full data is not published.

Q. Is the full data what appears in tab twenty-four?

A. That's right. I think so. Just a minute. That's right.

Q. I suppose perhaps the clear table then perhaps is table one at tab twenty-four?

A. I think so.

Q. Would I be putting it fairly that one of the conclusions of this study was the fact that...or was the proposition that amosite, as opposed to crocidolite, may have played a rather important role in the incidence of mesothelioma in the United States?

A. I think that's one of the implications here. I think one should also bear in mind the reservations that must be applied to this sort of study. I mean, in brief what it shows is that if you compare cases of mesothelioma with controls, and look for mineral fibers in the lung, dried lung tissue, of cases and controls, and you identify them as Dr. Pooley did, and you count them per gram of dried tissue, what you find is no difference between cases and controls in the amount of chrysotile, but you find an appreciable excess of amosite, and excess also of crocidolite, and then it fizzles out into small numbers.

Now, the reservation is, of course, to what

A. (cont'd.) extent does tissue examined at death reflect the exposure in life.

Q. What is the concern or what are the concerns in that?

A. Well, it is known that chrysotile is less stable in tissue than the amphiboles, so that no problem, we can say, right, this probably does reflect the history of amosite and crocidolite exposure fairly well, because this is very resistant, these are resistant fibers and they stay there, which of course could be a factor of why they cause trouble - they do stay there.

Perhaps the chrysotile has been dissolved out and we've lost the chrysotile effect. And I think one must still say we can't with conviction counter that argument.

On the other hand, there are a couple of factors which give you some confidence. One is that you'll notice that in fact there is very much more chrysotile in the lungs than amphiboles, so even if it has been washed out there is a good deal more chrysotile in the lung than amphiboles, both cases and controls.

The other point is, in another paper which I don't know if you've got as an exhibit, but it was another paper given at the same meeting by Rolands and wife?

Q. I don't believe so.

A. No. May I quote it?

Q. By all means.

A. In that paper, what they did was to take, confine their studies to chrysotile workers, men who had been working in the chrysotile industry who had left the chrysotile industry, and then followed up many years later when they died to see what relationship there was between the chrysotile concentrations in the lung and their work history. There was not a brilliant correlation, but a quite clear-cut relationship. In other words, the people who had the heavier exposure had more

A. (cont'd.) chrysotile in the lungs than people who had the light exposure.

So we have some support for thinking that when we compare cases and controls this probably doesn't simply reflect the disappearance of the fiber and the loss of the difference, but I don't think you can be absolutely sure of that and an awful lot more study is needed of this kind of problem.

But that doesn't, nevertheless, having said all that, that merely says, okay, we've no evidence in this study of any chrysotile effect in mesothelioma. It doesn't remove the fact that we have got a clear evidence of an amosite effect. As I said, there was a substantial excess in amosite.

And this is not too surprising having regard for the fact that amosite was very widely used in insulation materials in the United States.

Q. One other matter that appears from all of this is that the amphiboles seem to cause, more often than not, peritoneal mesothelioma, and just accepting for a moment that your cohort were in fact exposed to chrysotile, those workers cause pleural mesothelioma?

A. Right.

Q. Is there any significance to that?

A. There is one piece of significance, because I think that one of the things it does do is to say it looks as if chrysotile-related mesotheliomas are a bit different from the others. It looks as if they are pleural rather than peritoneal. Therefore, when I was saying earlier I don't know whether these cases in the mining industry are due to chrysotile, what was just described is some evidence that they are, because the very fact that they are of a certain type would kind of reflect that.

I have recently been studying a thesis which has a bearing on this, if I may again quote?

Q. Sure.

A. This is a study in the United Kingdom of all cases of mesothelioma known to the Cape Asbestos Company. Now, it so happens that the Cape Asbestos Company have, in addition to their crocidolite use, which has been extensive, have also had some chrysotile operations. The first point is that, again, the old confirmation - all the mesotheliomas are in the chrysotile. plant...sorry - crocidolite plant...very few in the others.

But what he also shows, and which I think is very interesting, there appears to be a relationship in this study between the heaviness of exposure and the site. In other words, the heavily exposed people have a high proportion of peritoneal mesotheliomas. The lightly exposed people, even to crocidolite, tend to have pleural mesotheliomas.

So what you could imagine is that there is certain potential for producing mesothelioma, that it takes a much higher dose of chrysotile, and that therefore in relation to dose, chrysotile very seldom gets, even if it causes mesothelioma, it much less seldom gets to the point where it causes the peritoneal one.

I think this is...it isn't anything more than, I think, if you like, speculation with some supporting data, but it could explain this. I think it makes sense.

Q. Has your research been able to establish any dose-response relationship with respect to mesothelioma?

A. I think other people have got later data than we have on this. For what it's worth, there was a crude relationship in our data, but other people have got evidence. In particular there has been the study done on the Nottingham gas mask workers, where they've got more cases and where they've made a careful study, and also in, again, the Cape Asbestos Company...maybe Mr. Berry mentioned it, but they have some

A. (cont'd.) evidence which is better than ours.

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.....to page 95

Q. What about non-occupational exposure and incidence of mesothelioma; are you finding any of that in your investigation -- are you finding non-occupationally exposed persons contracting mesothelioma, or, to put it more generally, a low dose ---

A. I think the preliminary point is, are all mesotheliomas attributable to asbestos? I think they fairly clearly are. So I think there's very good evidence that mesotheliomas antedate the commercial use of asbestos; that, almost for sure, there is a low base-line occurrence of mesotheliomas due to causes unknown in the general population, and probably one which affects men and women equally.

And if you look at the distribution of cases, what you find is that, as the rate for mesothelioma in a population goes up, so the sex difference increases, and so the proportion of mesotheliomas in that population associated with occupational exposure increases.

So that, if you have a population which has a high frequency of mesotheliomas, then you find there's a lot of males and a high proportion of the cases have got a history of asbestos exposure.

If you go to a low-level community, such as a lot of Canada is, then you find that men and women are not so different in numbers and you can't find any very good evidence of exposure.

Now, then if we come -- so what we're really saying is that -- and I may say the situation is changing very rapidly. The data which we have suggests that mesotheliomas in North America are increasing in number by between five and ten per cent per annum; right? Again, not surprising, because this would be what you would probably expect in relation to use of asbestos.

A. (cont'd.) Now, my guess is that most -- and the other interesting thing is, this is only in males; there's no evidence of an increase in females. And what I think we're seeing is the asbestos contribution hitting mainly men coming in and increasing.

Now, at some point or other, I suppose there'll be a balance between control of asbestos environmental conditions, so that even although, shall we say, more is used, better expo... -- better control may balance that.

And, again, Berry has made an estimate of this. He reckons that the thing is going to flatten out, I think, in about 1990, or something.

So the point I'm trying -- it's a long answer to you, that I think we have to acknowledge, first of all, that not all mesotheliomas, in my opinion, are asbestos-related. The situation will change in time and in place; there isn't any single truth. You've got to be much more sophisticated than that, if you're going to want to understand the situation.

My guess is that now, in industrial cities, and particularly places where there's been a lot of insulation and heat-trade work, you're going to have a lot of mesotheliomas, predominantly males and predominantly related to asbestos.

Q. Can you just help me; I'm not sure I understand why you say we're now -- with this five to ten per cent increase per annum, why we're seeing the asbestos contribution. Is this the longer latency period?

A. I think it's the thirty -- shall we say, twenty to fifty, yearly.

Q. Can I take you for a moment to article 26 -- tab 26 of that, which is one of the recent articles that you did, entitled "Malignant Mesothelioma in North America."

A. Yes.

Q. And at page 1653, first of all, are occupational groups A through E all occupational groups exposed to asbestos?

A. Yes.

Q. And what about F?

A. No. If I recall right -- I need to look it up, but if I recall F -- no, just a minute; I'm sorry -- if you really want an answer, I'll have to read it. I can't remember; F will be defined somewhere.

Q. I see; F, other listed jobs thought to entail asbestos exposure, and then a seventh group (G) of unlisted jobs.

A. That's right. In other words, F, yes, it is a category in which it was thought to campaign the possibility of asbestos exposure. These are essentially in descending order, and ---

Q. And G?

A. G is where they were not thought to be associated with asbestos.

Q. So that there were a hundred and one cases of mesothelioma in North America between 1960 and 1972 resulting from -- at least, so far as your knowledge carries you -- non-asbestos exposure?

A. Yes. We've got a better estimate of this situation in this paper which you've already quoted in this Banbury report, if I can find it. On page 4, this makes an analysis of the distribution of asbestos exposure in cases and controls. And our statistician in London has calculated that the proportion of cases that would be prevented by removing the occupational exposure to asbestos would be fifty-three per cent.

We go on: taking into account possible errors in the data, and uncertainties, we believe the true proportion, in

A. (cont'd.) 1972, might be as high as seventy per cent, and somewhat higher (about seventy-five per cent) in 1974, which is the year we're focusing on.

You will see from the way I've said that that we were tending, throughout this article, to say, when in doubt, we'll put it up. So I would guess that, in 1975 -- this is males -- that perhaps three-quarters of the cases in North America were associated with asbestos, in males; whereas, ten years earlier, it would have been much lower.

Both the absolute number of cases would have been lower and the proportion associated with asbestos.

Q. Would have been lower?

A. Yeah. Because the increase is due to the asbestos cases.

Q. You're not seeing any increase due to the non-asbestos cases?

A. No; quite the reverse. The females are just completely like that, and no evidence of any increase.

DR. DUPRE: Just on this matter, Dr. McDonald, if I may go back to an earlier study of yours, which is at tab 8, there's a table at page 361, where you record the death of females, from domestic exposure, in that three females were associated with domestic exposure from an insulation worker.

Can one take this as consistent with your more general hypothesis, that mesothelioma is associated with crocidolite?

THE WITNESS: Well, yes, I think you could; I think it is consistent with that.

On the other hand, it does also bring up another very important point, and that is that the one form of non-occupational exposure, which looks as if it is a significant risk, is domestic exposure in the house of an asbestos worker.

THE WITNESS: (cont'd.) When you were asking earlier, is there any -- what about non-occupational exposures, my feeling is that there's very little evidence of non-occupational exposure being related to mesothelioma, apart from this important category of the household contact.

And I think it is known, from environmental studies, that the dust concentrations in households can be very high and long-retained; the dust hangs around in furnishings, carpets, and so on.

And in our studies in the Quebec mining area, we do have some cases in domestic contacts with mine workers.

DR. DUPRE: And these are cases of mesothelioma?

THE WITNESS: Yes.

DR. DUPRE: And I take it from this same page 361 that, when you did this early study, the '73 study, that that one case that appears on domestic mining and milling would be one such case?

THE WITNESS: Yes, it would. I don't know -- yes, it would. Is that stated to be in the mining ---

DR. DUPRE: Well, the table -- the lefthand side of the table says "Mining and milling." And then, under "Domestic," there's one case.

THE WITNESS: Yes; that's correct.

DR. DUPRE: Have you made a study showing more such domestic cases?

THE WITNESS: Yes. Indeed, it's important to note here that, when we talk about the eleven cases in our cohort, that that is not all the cases in association with the mining industry. The cohort is only roughly half the employees of the mining industry, and we have got other cases who are not in the cohort.

On the other hand, what we haven't got -- and we

THE WITNESS: (cont'd.) looked very hard for these -- is mesotheliomas in the mining region of Quebec; the only exception being cases in the families of miners.

MR. LASKIN: I want you to go to tab 26, page 1653 and 1654, because I think you deal with precisely those two points.

THE WITNESS: I'm sorry; which paper is it?

MR. LASKIN: Tab 26, page 1653 and 1654. This is the same article, on malignant mesothelioma in North America. 1653 and 1654.

Q. I take it you deal with, first, home exposure and then neighbourhood exposure?

THE WITNESS: A. Yes; two male and six female cases. They were not all mining cases, I don't think. Oh -- over the page, yes; three cases and one control.

> "In 3 cases and for 1 control, the clothing was that of a Quebec chrysotile production worker ..."

Q. And then, coming on to neighbourhood exposure, is that -- is the reference there the subject you were relating to the Chairman just a few moments ago? That is, you looked in the area of Quebec and couldn't find any neighbourhood exposure.

A. Yes. Well, I think we've had some -- I can't remember our numbers; we also had some controls.

May I also quote another small letter we wrote to a medical journal fairly recently, which I think has some bearing on this; and I think it could have importance, because it has been suggested that possibly children are more susceptible than adults.

Q. Well, I was going to ask you something about that.

A. I don't think we've got any evidence that they

A. (cont'd.) are, but, nevertheless, this may be relevant. Here we have it.

This is a letter to The Lancet on November 17th, 1979, in which we were commenting on the question of -- somebody had written an article about there being some relationship between latency -- the latent period and age, and I can't remember now the argument exactly, but I could go over it.

But one of the things we point out is, if I may quote:

> "We have some evidence that indirect occupational exposure in the home may account for some of these younger cases.
>
> "In epidemiological inquiries based on cases ascertained through pathologists, five were in persons whose fathers had been asbestos workers. Death in these five occurred at age thirty-one, thirty-two, forty-two, forty-two, and fifty-one years."

So the age was tending to also point to the importance of the childhood exposure in the household of an asbestos worker. I don't know whether all these were Quebec cases; I'm not sure they all were. But they were -- most of them were.

Now, that doesn't throw any light on whether there is increased risk in childhood; that merely says, there were cases in children and these -- I mean, that is to say, there were cases in young adults who had the opportunity of exposure in childhood.

Q. I take it -- because it is a subject which is of some concern here, particularly in relation to children being exposed to asbestos in the schools, do I take it that -- well, let me ask you: are you aware of any evidence, one way or the other, on the susceptibility of young children?

A. No. And I think I've seen in your list that you'll be seeing Mr. Julian Peto later, and I think Peto has been making a study of latency in mesothelioma in relation to age and has found no relationship between age and latency.

Now, that does not mean that there's no difference in susceptibility, but it would tend to suggest there wasn't, because, if there were, you would probably get some change in latency, or might do.

Q. You'd expect to see it earlier?

A. You might; and he didn't find any. But he can probably answer that better -- I mean, answer it better than I can.

MR. LASKIN: I've got about one more topic to cover, Mr. Chairman, and I don't know whether you want to take a quick break now for about five minutes.

DR. DUPRE: Just before we do, I was wondering, as Dr. McDonald's last remark washed over me -- I just want to make sure that I understand the point that you made.

The point that you made, does it boil down to this, Dr. McDonald; that there is no evidence that you know of, from epidemiological studies, that would indicate that the lungs of young persons are inherently susceptible to asbestos-related disease?

THE WITNESS: I prefer to confine the observation to mesothelioma, because I don't think the lung cancer and asbestos question is really a realistic question in relation to children.

But, obviously, the mesothelioma issue is a question of possible importance. On that, I know of no evidence that children are more or less susceptible than adults. I suppose the evidence tends to suggest that they are, so far as it goes -- are equally susceptible.

DR. DUPRE: Shall we break until twenty past 4:00?

MR. LASKIN: Twenty past. Thank you, Mr. Chairman.

INQUIRY RECESSED

.....

INQUIRY RESUMED

MR. LASKIN: Dr. McDonald, can I turn to another topic, just briefly, and it's really the topic of ordinary environmental exposure.

Now, I know that your own particular research was in the occupational field, but I wonder if I might draw on your professional expertise for just a moment.

Q. Can you help us at all on what we are looking at by way of the magnitude of the health risk to the public, for example, by way of exposure in problem buildings, to use a term -- for lack of a better word?

What kind of a health risk do we have in schools that may have loose asbestos and involve exposure? Can you help us on that kind of subject?

THE WITNESS: A. I mean, it is certainly more than schools, isn't it?

Q. Yes.

A. Hospitals and public buildings.

I suppose my approach to this is, this is a prize example of the necessity to establish the exposure-response relationship, because there's no way you're going to be able to directly measure hazards associated with the types of level of exposure which are sort of casual in that way.

Now, I suppose you'll have gathered from my comments so far that I am very concerned about the capacity of the

A. (cont'd.) amphiboles (in particular, crocidolite and, I suppose, by extension, probably amosite) to cause trouble as a result of relatively low exposure; and, in particular, mesothelioma.

So I suppose that I would take a rather different view of any casual environmental -- maybe regular, if it's a school -- exposure which was to measurable amounts of amphiboles.

I guess there's still -- the principle, however, is, if you want -- well, I mean, one very good principle is that whatever we've done in the past, it's obviously clearly crazy to use asbestos for things you don't need to use it for; for example, children's modelling clay has been a case in point, which is -- you know, could easily -- there's no point in doing it, but it was done, or women's fur coats, or whatever.

But if we put that on one side, then it seems to me what you're doing is extrapolating from exposure-response relationships, which you're only going to be able to obtain, in my opinion, from occupational studies. And they're difficult enough there to get them, and therefore it is a matter of doing environmental surveys in places to find out what is the level of exposure, and working out, from that, what the risk is.

But I say, I would feel that any form of ... From what we know, I don't believe it would be tolerable to allow uncontrolled exposure, even at a very low level, to crocidolite; I just wouldn't be interested in looking at the exposure-response relationships. I'd just say it's just not worth trying to do.

But it's another matter when you come to chrysotile; and here, I think that I would simply apply the data that one has, bearing in mind, again, that one doesn't want to use the stuff if you don't have to use it, for non-essential

A. (cont'd.) purposes. There are plenty of semi-essential purposes without doing that.

And if that comment helps -- perhaps it's too vague.

Q. Applying the data that we already have and, I take it, extrapolating, can you give us any more -- I hate to use the word "precise," because I know one can't be precise in this field -- but can you give us any more definite assessment of what kind of risk we're looking at to the ordinary public?

A. Can I, for the purpose of this discussion, make no further reference to amphiboles?

Q. Sure. I think we understand your position.

A. I mean, I just don't think it's worth getting into that.

Q. That's fair enough.

So let's talk about chrysotile.

A. Chrysotile.

Now, if you start discussing chrysotile, you've then got to face up to the fact that quite a high proportion of the earth's surface is made of serpentine rock, and quite a lot of it is crystallized and quite a lot of chrysotile asbestos is everywhere, and it isn't necessarily the result of industrial activity.

So chrysotile is very common; it's common in water supplies, it's common in rain, it's common in all sorts of

A. (cont'd.) things.

So, I suppose the social decisions which will be made will at least take into account background levels, because it might be that we would also want to clean up the world we live in -- if you like, the natural world we live in -- but I guess we're now talking about man-made pollution.

So I think it is a matter where -- I mean, I'm not just being academic here; we're often asked to go and measure the concentration in schools and in theatres, and so on, in order to find what is the level, and then, I mean, it's a matter of looking at what you know about exposure.

It does seem to me, for example, that in one form, big, public entertainment places, I believe it is too far-fetched to think that there is any hazard to the people who go to see the theatre twice a year, or something. There is some hazard, conceivably, to the regular employees.

Having seen the -- I find it still difficult to take very seriously because of the levels being so much lower than the lowest occupational exposures recorded; but the snag about it is that you've got an awful lot of people.

You see, I mean, one of the things that we -- if you agree on a linear exposure-response relationship, you then have to start saying, well, the risk is only one in ten million of trouble, or a hundred million of trouble from, say, going through a tunnel in the London underground railways; but then you say that there's about a hundred million who do that every day.

So, in terms of the impact on society, I suppose it then might be that, as a result of going by train, there is one more lung cancer a year in Britain than there would be otherwise, you know, or perhaps more than that; I've no idea. It's this kind of thing.

A. (cont'd.) And I do think that you can't really discuss this kind of question, again, except in the field of social awareness and concern.

You see, it seems to me so totally unbalanced to consider that sort of risk in relation to what is probably an infinitely bigger risk from sitting next to somebody who smokes a cigarette -- and I don't mean smoking it yourself.

MR. LASKIN: We'd better keep the Chairman away from you. [Laughter.]

THE WITNESS: You see, I mean, passive exposure, all sorts of things go on; but I think these are social decisions. If you want a scientific estimate of risk, you measure the concentrations and you apply your dose-response relationships, and that tells you what you'll expect. And there's no better scientific answer than that.

MR. LASKIN: Fair enough.

DR. UFFEN: Dr. McDonald, do you know of any organized studies, published or unpublished, about background levels. Suppose we wanted to establish what is a normal background level; do you know of any?

THE WITNESS: I know that there are such studies, and I'm afraid I'm not familiar with them; I'm sure one could find out the references, because there have been quite a number.

MR. LASKIN: Can I just turn to the last topic that I wanted to discuss with you, and I'll try and do it briefly, because I know I've kept you here for a long time; and it really is the question of detection, early detection, of evidence of asbestos-related disease and, secondly, the effect of removal of persons, employees, from asbestos exposure. And I guess there are two questions that I really wanted to put to you, and perhaps you could discuss them with me, generally.

Q. First of all, what is -- what is or what are

Q. (cont'd.) the most sensitive indexes, or early detectors, of asbestos-related disease; and, secondly, what does your own research tell you about the effects of removal of workers exposed to asbestos on their future health?

THE WITNESS: A. I take it we're now referring to occupational exposure?

Q. Yes; I'm coming back to occupational exposure.

A. Occupational exposures; and I assume that we could put on one side what is the earliest method of detecting a lung cancer or a mesothelioma on the grounds that that wouldn't be awfully useful; I don't know.

Q. Well, fair enough. Let's talk about pulmonary fibrosis.

A. All right; pulmonary fibrosis. Right.

Well, I think what you have here is the problem that the earliest method -- that you always have a conflict in all tests, between what we call sensitivity and specificity.

If you have a sensitive test, it will not be specific; so that if you look for very small deviations from normal respiratory functions, or small deviations from the appearance on the X-ray -- and those are the only two methods I can think of offhand that you've got -- if you calibrate those very, very carefully, and if you ... You see, the main problem in all these methods is that you're looking for very small changes, and therefore you must have extremely high technique, because there is more variation between two X-rays of the same person, commonly, than there is between an asbestotic and a healthy person. You get that degree of variation simply from taking the picture.

So, if you're going to use these methods for early detection, they must be very highly standardized, so that the quality of radiology is very high, the level of reading is very good. These are constants of perfection. Then I think you have

A. (cont'd.) the earliest means of detecting deviation from normality.

And, again, the detection will be most sensitive in groups and not in individuals. You'll do better by studying a hundred persons exposed, and you won't be able to detect which one's the abnormal one, but the group as a whole will give you the most sensitive indication that that group is having more trouble than another group, you see.

So I would say -- but having said all that, we then have -- we've manufactured the most sensitive approach we can, but what we then have is to say, but is it anything to do with asbestos, for example?

We have created a very sensitive method of detecting departure from what it was, but then it could be due to something else, because the changes which you're looking for are not specific for asbestos.

There are, really, when you get down to it, almost no tests that are specific; I can remove my qualification -- I don't think there any that are specific for asbestos, as such.

DR. MUSTARD: Can I just pursue this a bit further with you.

In the tab 24, which is the McDonald-Pooley paper, there's a dilemma for me; that one of our witnesses said it may be possible, using modern techniques (fibre optics, et cetera), to explore down into lungs, which, of course, could allow you to get direct sampling of material from the lung which might indicate that there are asbestos fibres present and might also indicate that there are any changes of fibrosis taking place by observing some of the fibreglass responses.

But as I look at the table 1 in that paper, and we'd have cases versus controls in the mesothelioma story -- I realize this is not for asbestosis -- but I suspect we'd have a

DR. MUSTARD: (cont'd.) bit of an interesting dilemma, diagnostically; that you could have a person with asbestos fibres who might have a mild tissue reaction not sufficient to produce clinical changes in your sophisticated test but, nevertheless, biologically, if you could measure that point having effects; conversely, you could have a person with asbestos fibres and have changes in your pulmonary function test.

Are we really into a catch-22 in trying to become enormously specific about this, in that, in a sense, at the present moment, we're restricted to the asbestos exposure story and the X-rays, which have enormous limitations in terms of specificity, and, as you said, the limitations of pulmonary function tests; so that we're caught in a situation where you're using a conventional diagnostic based on your technology now.

If our technology improves, we can perhaps detect the earlier changes in fibrosis, but we'd be caught with the dilemma as in table 1. Is that not part of the problem? Am I being too simplistic?

THE WITNESS: I followed your argument entirely, apart from table 1. I am unable to relate your story to the table.

DR. MUSTARD: Well, we've seen a few other tables as well, and my problem has been, and I hope we'll get more information, that when you take lungs and extract them, you find asbestos fibres in them. And it doesn't matter what you seem to have died from, there are asbestos fibres present. Those people have died at the age of sixty and seventy without a diagnosis of asbestosis -- they may have a small amount of fibrosis -- and my dilemma is, as you probe deeper into the story, you may find, indeed, there are people with asbestos fibres in their lungs and mild amounts of fibrosis that never really reach clinical significance, whereas there are others that do, and the amount of

DR. MUSTARD: (cont'd.) asbestos fibre in the lungs may, indeed, be possibly the same, which makes it a very tough problem to sort out.

THE WITNESS: Oh, I think that's quite true. On the other hand -- I mean, before sort of leaving table 1, I think this is an example, if you like, of rather the reverse; that is, there's a high degree of specificity.

We have here the counting of very highly specific changes; namely, fibres which are identifiable -- countable -- and a comparison, a straight comparison, of numbers between cases and controls.

DR. MUSTARD: Has this been done for asbestosis?

THE WITNESS: No, it hasn't. There's a vast field of research open here; yes.

DR. MUSTARD: I just sort of felt that chrysotile causes asbestosis, I believe.

THE WITNESS: Oh, yes.

DR. MUSTARD: And I just sort of wondered if you got into that kind of degree of controls showing that much contamination with asbestos.

THE WITNESS: I think -- well, this is where you have to, of course, pay considerable attention to the design of the survey, to see that it's properly controlled.

So, I mean, the interpretation of findings in cells, or the case of asbestosis, would depend very much on what you were comparing them with.

And I appreciate that the diagnosis of asbestosis is a good deal more woolly than a mesothelioma, which we can be more precise about. So I do agree about all that.

Indeed, if I can say that there have been some studies already which, in my view, are extremely misleading, in which my good friend Pooley did the technical work, because it

THE WITNESS: (cont'd.) illustrates how good technology can be -- give you totally meaningless results, if you don't design it properly.

I mean, what he did, he was served up with a whole lot of lungs of so-called asbestotics from the Quebec industry; no controls. And what he found was, to everybody's astonishment, that the thing was full of tremolite -- these lungs were full of tremolite.

His colleague, who was less critical than some, proceeded to advance the hypothesis that maybe chrysotile isn't the cause of the asbestosis; it's the tremolite.

I really think that that is the kind of thing where you've got a test where you measure something, but you can't -- what we're looking for is a design which will allow you to compare.

So my feeling is, the technique here of a highly specific test is a jolly good test, but its application to early detection -- I don't see a way at the minute.

But what I was worried about was, the tests we do have available, if they are to be sensitive, are very non-specific, and, therefore, I don't know what you do with them.

What I think, in the end, the protection of the individual in an occupational condition is really the maintenance of control on the environment, to my mind; and it is a matter of proper monitoring of the environment, using human tests as a sort of back-up.

Because, after all, you may have your own ideas that a certain level of fibres is safe and everybody is quite all right, but it's rather desirable that, every now and then, you look at the workers to see if perhaps they're not all dying of something, you know, because you've got to validate it.

My feeling is that, in fact, throughout

THE WITNESS: (cont'd.) occupational medicine, screening of workers is of very limited value, very limited; but as a means of back-up and a validation of the environmental test it's useful.

One of the things we don't have, actually, in asbestos -- I was very interested in a technique which was being tried out in the Quebec mines, and I haven't heard the outcome, was a method of being able to quantify stored asbestos in the lung by a magnetic process, so that you simply had a method of measuring, in life, how much accumulated fibre there was.

Now, I don't know -- I mean, if you could imagine that kind of thing being developed so that it did give valid results, this would be a form of biological monitoring which was not looking for disease; was simply looking for what you might call normal storage. Is this person accumulating any detectable amount of dust in the lungs?

But I don't think we have that for asbestos workers at the present time, and, at the moment, I really do think we're in this sort of catch-22 position, which you referred to, that we've got methods which, far from helping us, are positively discovering, more and more, less important things, and what do you do with them?

But you also -- did you ask me, or were you going to ask me, the question of removal?

MR. LASKIN: Yeah; that's the second question. And I know that you and your colleagues have done some work on that, and perhaps I could take you to tab 16, which I gather is the paper that deals with that question.

Q. As I understand it, you looked at a cohort of eighty-six men who left the industry between 1950 and 1961 and who had a withdrawal X-ray within twelve months of leaving and then had no further exposure to asbestos.

Q. (cont'd.) And, as I take it, one of the principal conclusions of this study was that 9.3 per cent showed an increase in irregular small opacities, which I take it is evidence of asbestosis, after withdrawal.

Can we draw any conclusions -- what I'm troubled with, can we draw any conclusions from that as to the effect of removal from the work place?

THE WITNESS: A. Not really a lot. I mean, the conclusion I draw from the study -- and I have to confess, at another time, I tried to design a better study, because I think it is an important subject, and I don't really think -- it happens to be, I think, the only one there is, but there are lots of reasons for not being happy about it.

What we -- I mean, even taking at face value the fact that there was progression, I think it's a fair conclusion that it's not one that will surprise anybody, because it is well known that there is a delayed effect from exposure, and therefore it would be surprising if people did not progress after leaving exposure.

What we don't know is whether they progress more or less than they would have done if they had gone on being exposed; we don't know that.

Q. That was my -- that was what I was really getting to; I couldn't get from this paper any indication as to the answer to that question.

A. No; I think you're absolutely right. I don't think it does answer that question.

Now, I think in our discussion somewhere we do sort of confess a few things; next time we'll do it differently.

It also illustrates another problem, if you like, which we were referring to just now: the importance of technique. You see -- I mean, the essence of any such study depends upon the

A. (cont'd.) X-rays taken, shall we say, twenty years later, being entirely comparable to the ones that were taken before you leave, or they weren't; we know that. And, therefore, we could only interpret quite gross changes, meaning, I think, that they had to be big enough that we'd say we don't think they could be due to technique.

No; I think what it does, for what it's worth, is to confirm what is often believed; that the disease does progress ---

Q. Even in the absence of exposure?

A. --- in the absence of exposure, and it does confirm, therefore, the view that, if you want to know, if you like, what is the effect of exposure, and you go in and examine a work force now, it probably is underestimating the effect, you see, because you're now looking at it at a certain point in time.

Now, probably five or ten years later, even if they had been withdrawn, they would probably have a higher prevalence of change than they had then. So that, perhaps, is useful.

But as a test of, is withdrawal useful, I don't think we know. Once again, I'm quite sure that one has to take into this story, because it is, after all, a practical point that you're bringing out now, is it a good thing to withdraw workers who show changes?

I think it obviously is, from a social and practical point of view, very important to consider the age of the worker and what his present conditions are.

I well recall, if I may say so, a few years ago when there was a lot of concern in the Quebec mines and mills, that one of the physicians concerned was very seriously criticized -- I mean, most unpleasantly criticized -- for allowing workers to go on being exposed. Now, these were men who perhaps were fifty and sixty years of age, for whom, almost for certain,

A. (cont'd.) whether they went on working or not, made no difference to their health outcome, and yet it made an awful lot of difference to their happiness and their income.

And I do think one has to consider the social aspects of withdrawal in addition to what you might call the purely medical ones.

On the other hand, what is most important, of course, is not so much the withdrawal of the man, in my opinion, but the recognition that changes are occurring and, therefore, action is needed.

If changes are occurring, obviously environmental control is not adequate, and, therefore, further stricter control is needed.

And then I think what you do with the young worker is something that needs to be discussed very carefully with him, and in relation to what control is feasible. I mean, if you can strengthen the controls and say that this environment is now back to what should be a reasonably safe condition, I would think there wasn't a strong case for withdrawing the man, unless he said, "I don't like this job; I want to get out of it." He should be taken into account, in other words.

Q. Fair enough.

Is the timing of when you withdraw a person -- could that be important in relation to the progression of the disease?

In other words, what I'm thinking of, is there a point in your lung which progression's irreversible, regardless of what you do?

A. I don't think -- I don't know of any evidence, and I must say I'd be surprised if there were any -- any particular point.

It could be that the more advanced the disease,

A. (cont'd.) the less relevant withdrawal is. And it's this old business: if you're already bad, you can't get much worse; whereas, if you are not very bad, you have the whole range of progression possible.

Q. Is there any ongoing work of which you're aware in this particular area?

A. I think that, in Britain, there is a register of asbestos workers, and the government has not decided that the economy can't continue to maintain it. I think they are intending to keep on observing the workers and observing what, in effect -- it would include things like this; what happens if they're taken out or transferred. Certainly, the mechanism exists there for evaluating this.

MR. LASKIN: I'd like to thank you, Dr. McDonald; I think I've run out of questions, and I'd like to thank you for bearing with me all day.

I think, Mr. Chairman, I certainly am finished. As I said, I canvassed with my friends the situation with respect to their questioning and, without pinning them down -- and I've indicated to them our timing problems, and they have timing problems themselves -- they've given me some assurance that, if we start at nine o'clock tomorrow morning, and if that's acceptable to Dr. McDonald, that we would finish in time for everyone, including Dr. McDonald, to make their respective travel arrangements, and so on.

DR. DUPRE: Well, of course, given Dr. McDonald's timing, four o'clock is the absolute tomorrow. At that point, we will all turn into pumpkins.

THE WITNESS: I'm perfectly happy up till 4:00; if you'd like me to stay longer than 4:00, I do have to make other arrangements, which I can do, but ---

DR. DUPRE: I think we all wanted you to remain

DR. DUPRE: (cont'd.) perfectly happy throughout, Dr. McDonald, so four o'clock it will be.

Now, Dr. McDonald implied to me, at the coffee break, that he would be willing to have someone, say, open up, to go, say, till 5:30, but not much later than that; it's about eleven o'clock his time.

Would one of the parties like to open up a line of questioning that, of course, could be continued tomorrow morning at 9:00 a.m.

DR. NELSON: That's fine with us -- I mean, you know, depending on how Dr. McDonald feels. If he's tired ---

THE WITNESS: No; I'm not tired at all -- tonight is ---

DR. DUPRE: Would you now start, then, Mr. Nelson?

DR. NELSON: Sure.

DR. DUPRE: If you please, then.

THE WITNESS: Is this now -- excuse me, Mr. Chairman -- is this now a kind of an informal discussion, or is this ---

DR. DUPRE: No; the -- [laughter] -- is that it's continuing. You had lengthy questions from one of your students from 10:00 to 5:00, and now we have a second one, Dr. McDonald.

CROSS-EXAMINATION BY DR. NELSON:

DR. NELSON: Okay. Well, I guess -- it seems to me, the purpose of this whole meeting is to develop an occupational asbestos standard for the Province of Ontario.

Q. And I'd like to start, Dr. McDonald, by asking you what you would regard as an acceptable standard -- occupational standard -- of asbestos for the Province of Ontario.

THE WITNESS: A. I can certainly say, straight away, that I would never be prepared to answer such a question. I don't believe it's my job to.

A. (cont'd.) I would -- the kind of things that I would suggest should be taken into account, however, are that I would urge that any legislation for controlling environments should recognize what, to my mind, is very clear; and that is that different types of asbestos have very different risks, and that, equally, there is strong evidence that different processes have different risks.

And, therefore, I think the issue would then be whether, socially speaking, if it is acceptable in the Province of Ontario to legislate everybody to the most hazardous material and the most hazardous industry; because, obviously, that could have enormous repercussions in industries where such standards are not necessary.

So, I mean -- those are the kind of things I -- the second kind of thing I would point out are the -- what you might call the importance, on the one hand, of exposure-response findings, where they exist, and the overall principle that it is ridiculous to, in my opinion, regulate for processes that are unnecessary.

Q. I realize it's a difficult question. I guess I'm trying to get a sense from you, for instance -- like, a ballpark figure, if you feel that the two-fibre standard is -- would protect workers adequately, or if you would subscribe to the recommendations, for instance, of the NIOSH-OSHA asbestos work group, April, 1980, where they suggest one-tenth of a fibre per c.c.

I mean, I'm sure you have some feelings as to whether you feel those standards, one or the other, is too high or too low. Can you give me a sense of that?

A. Well, if I thought any workers were going to go on being exposed to two fibres per c.c. of crocidolite, I would be, obviously, extremely unhappy. In fact, I don't know

A. (cont'd.) that I'd be all that happy if I thought they were being exposed to point one of a fibre of crocidolite. That's a different matter, isn't it?

And then I think I would then have to throw it back at you, I suppose, and say, what do you and your colleagues feel is the sort of measure of hazard which you think is acceptable; because there isn't any safety, you know. I mean, I think this has to be pointed out. There isn't any safe level of asbestos; there isn't.

Q. We agree with that last statement.

A. Or anything else, I should add.

Q. Well, that I don't agree with. [Laughter.]

A. Well, if you can think of anything that's safe, I'd be interested.

Q. Well, I would like to get into that, but maybe tomorrow. I think there certainly are differences in terms of exposures to different substances; I don't think there's much doubt about that.

I guess one of my concerns is the submission to the Royal Commission on Matters of Health and Safety Arising from the Use of Absestos in Ontario, the brief that I believe **they** submitted to this Commission -- that's from the Quebec Asbestos Mining Association, January, 1981.

On page 19, they refer to your study, which is article number 18 in the booklet, regarding dust exposure and mortality in chrysotile mining, 1910 to '75; and, in referring to that study, they refer, in essence, to the last paragraph of your abstract, in which they say, thus, it would seem that if it's almost impossible to discern an increase in risk at an exposure rate of twenty fibres per c.c., it is all the more so at -- then they say -- two fibres per c.c., which is the present standard for dust levels in the asbestos industry.

Q. (cont'd.) It seems to me, what they're concluding from the last paragraph of your abstract is that the two-fibre-c.c. level is a safe level, and that they reach that conclusion from your previous sentence in the abstract, about finding no excess mortality due to cancer at the twenty-five-c.c. level. Could you comment on that?

A. I think it illustrates that any sentence taken out of context can be misleading, and, therefore, it is obvious that the sentence does not reflect any part of the conclusions of the article.

I mean, the main theme of the article is that the exposure-response is linear.

Q. So you would not agree, essentially, with that last paragraph on their page 19, in which they deduct from your paper, it seems to me, that the two-fibre-c.c. level is okay, on the basis of your paper?

A. I haven't seen this, but it's unlikely that I would agree with that interpretation; but, then, I don't know what we mean by "okay."

What do you mean by "okay"?

Q. That they would -- that they feel the current standard of two-fibre per c.c. is -- should remain; let me put it that way -- that it adequately protects workers.

MR. WARREN: Mr. Chairman, let me ---

THE WITNESS: I can't interpret the statement.

MR. WARREN: --- interpose, if I might, a suggestion here.

I think it's very difficult for all of us out here to deal with questions which quote from, or purport to quote from, a document which we neither have, nor does the witness have. I think it's awfully difficult for the witness and for the attorneys to come to grips with both what is being asked

MR. WARREN: (cont'd.) and what is being responded to, when we don't have the document on hand.

DR. DUPRE: Well, we are going to go on at nine o'clock tomorrow morning, so perhaps the particular brief, the page of the particular brief that is being referred to, could be made available to everyone during the break.

MR. LASKIN: I'm sure it can; but I certainly echo Mr. Warren's sentiments, particularly as it relates to the witness. I think it's rather unfair to the witness to put to him a summary of a statement that he hasn't seen.

DR. NELSON: I'm sorry; you know, I've never been to one of these hearings before. I assumed that everybody had this; I mean, it's a brief submitted to the Commission. And, you know, fine; I mean, I have no objection to people reading it. I assumed that it was in evidence, or whatever you say.

THE WITNESS: I mean, I can make a general statement that might be relevant to this, and that is that I think that the results of the cohort mortality study should, so far as possible, be understood by the people who are concerned, and obviously that means government, the employers, and the workers; and, to this end, we had a full-day meeting at Thetford Mines to discuss with the unions this paper in detail, in which we discuss what linear relationships mean and what risk means, and so on; because I do believe that this is what counts, that the workers know what you -- or, so far as is possible -- I mean that, so far as possible, we need to emphasize -- and I really did mean it -- that there is no such thing as a safe environment; that some things are more dangerous than others, and a few things, we've actually tried to measure them, of which one is asbestos. And that there are data on this, and this is the date.

And I think it's very important that workers and

THE WITNESS: (cont'd.) employers should understand what that means. Now, after that, it's certainly not for me to say, you should tolerate a one-in-a-thousand chance of getting lung cancer; it's not for me to say that. I can't say -- I think, in my view, they have a perfect right to say, "Yes, we'd be glad to tolerate that," if they know what that means.

DR. NELSON: Right. Well, I think that's why we're all here and, you know, our aim is to protect working people, and the whole question is, of course, what is a safe level and what is not?

I guess, in preparation for tomorrow -- I don't know how this is done, but I would like to be able to distribute to people here (whoever is in charge of that) this report, this brief, that I believe was given to the Commission, and a proposed regulation on asbestos, I believe, given to the Ontario Ministry of Labour. It's by the counsel for the Asbestos Information Association. I'd like these two submitted, if that's okay, so I may refer to them tomorrow, and we'll do that then.

MR. WARREN: Just to make it absolutely clear, Mr. Nelson, I have no objection to that way of proceeding at all; simply that the matter is, when we don't have in front of us, as we've had with one or two other documents, the text, I think it's hard for us and doubly hard for the witness, and it's on that ground, and that ground alone, that I interposed the suggestion.

DR. NELSON: Sure; I understand completely. I assumed people had them.

THE WITNESS: It would be helpful to me, too, if things like the term "safe" were defined.

DR. NELSON: Q. Well, let me put it this way. I guess what I'm trying to ask is what you regard as an acceptable

Q. (cont'd.) standard. That's why we're here.

THE WITNESS: A. Myself?

Q. Yes; yourself.

A. You mean, if I were -- the sort of standard of safety I want in my work?

Q. Well, you hold a position as a physician and a scientist, where you've studied asbestos exposure, and you've made various assertions in a number of your articles as to what you feel the evidence indicates and the reason you've been called here today, I believe, is to -- so we can all learn together about what we feel an acceptable exposure level is for working people. And that's, I think, what we're trying to find out, and that's why I ask you the question.

There's no doubt in my mind there will be a conclusion to this conference and that a level will be determined, and we hope that it's a level which protects workers. And the only way that we can determine that, in the course of these meetings, is to ask people such as yourself, who are before us, what you feel is a level that's acceptable, or safe, and levels that you don't.

A. Well, I would like to -- I think it may save time later if I, here and now, categorically say that I don't believe I can ever answer that question. I don't believe I should answer that question; I think it would be immoral for me to try to answer it, and I don't propose to try to.

I am very happy to try to define, as best I can, the risks associated with work. What I can't do is to tell people what they should do. I don't -- this is one of the qualities of expertise that doesn't exist; I mean, experts do not know what is acceptable to other people. And, therefore, it would be -- you know, this disposes of the question, as far as I'm concerned.

A. (cont'd.) I've written an article to this effect -- I don't know if it's in your text -- it isn't a unique point of view; it is absolutely universally held in most of Western Europe -- I don't know whether it is in North America -- the very concept of asking a scientist to define an acceptable risk would be considered absolutely immoral, where I come from.

Q. Okay. Well, if I can continue, I wanted to ask you about the second paragraph of the abstract, of article 18; I believe you do have that, and that the people on my right have that.

In the second paragraph of that abstract, you say:

"By the end of 1975, 4463 men and 84 women had died. [That among the men], the overall excess mortality [from 1926 to '75], was 2% at Asbestos and 10% at Thetford Mines ..."

Now, I'd like to know what your feeling is about that overall excess mortality; how serious you feel that excess is, and how significant you feel that is.

A. Well, in the statistical sense, it's highly significant; in the social sense, I think it's important.

I think that any occupation which raises the excess mortality, from all causes, by ten per cent is quite, in relative terms -- quite a high risk.

I say this, because you appreciate that asbestos doesn't -- is not a cause of all causes of death, and, therefore, it means that, whilst the ten per cent is increased, that that probably is concentrated in certain causes of death. And, indeed, we've seen that it is.

It's concentrated in respiratory diseases, in cancers. There's some overlap into other causes, but this is where it lies.

Therefore, it means that, in those causes, the

A. (cont'd.) excess is bigger than that; it has to be. So it also, of course -- there's a big limitation in the use of the index we're using, which is the normal one to use (the standardized mortality rate) -- this is all men, and not all men are heavily exposed; in fact, most men are not heavily exposed, even historically.

So that if we then look at the experience of men who were heavily exposed, of course, this is a considerable understatement. That is one point.

On the other hand, it is not misleading in that I think it's always very important to keep -- I mean, it is, I think, very useful to say, okay, there is, let us say, in heavily exposed asbestos workers, not ten per cent but three hundred per cent of their normal expectation, let us say, of lung cancer, and that sounds, of course, very, very serious, which it is.

But it still needs to be put in the perspective of -- because, for the worker, he's not so interested in what is put on the death certificate; he's more interested in what his prospect of living is. And, in relation to that, this figure gives him some sort of picture, that he has a ten per cent increase in that industry, overall, bearing in mind it'll be worse in the heavily exposed, less in the lighter exposed.

So, in other words, any kind of summary figure like this is useful, but it has its limitations. I think it also is useful to look at the difference between Asbestos and Thetford Mines, because that is not -- the two per cent doesn't sound very impressive, and, of course, one of the -- I haven't had time to go into this, either -- the very concept of a standardized mortality rate, it means compared with the general population.

Now, the problem really is, are the workers of

A. (cont'd.) Thetford Mines comparable to the general population? In some ways, they are; in some ways, they aren't. And so it has that limitation; you know, it is compared with Quebec as a whole; it's not compared with the immediate counties of -- you know, which might give you a slightly different answer.

Another point is, you are probably all familiar with an expression that's had a lot of use recently (the healthy worker effect), which is a reality; that is to say that, in general, people who are employed have a more favourable health experience than people who aren't employed.

For obvious reasons, if you're sick or unemployable on account of sickness, you have a higher mortality; therefore, the SMR here tends to underestimate the impact of industry; there's no doubt about that. Tends to -- not in every industry.

There are situations in which there is selection of more less healthy people into an industry, but I don't think that generally applies to the heavier industries like mining; okay.

So let's say, straight away, that those figures perhaps underestimate the impact, but still it is -- I must say, I personally was struck by the fact that Asbestos, which certainly, we say here, "...Thetford Mines, much the dustier region." I don't think anybody would question that, who has seen it or seen the measurements.

But the Town of Asbestos, and the mining in Asbestos, which has gone on for the same time, is by no means a paragon. The dust counts that this cohort in Asbestos had worked through were very high. Let us say they were the average in 1950, in Thetford Mines -- I've got it written up somewhere; it was something in the order of twenty-five-million particles per cubic foot, which probably means of the order of fifty to a

A. (cont'd.) hundred fibres; and yet we've only got a two per cent excess mortality.

What I personally feel is terribly important is that -- why I emphasize the point of different industries, I don't think there's anything misleading about this date; I'm quite convinced now that it is the truth; it's so consistently found now, that it's fairly evident to me that safety, at least to the level that is acceptable to workers -- and, having talked to them, I believe this is true -- is achievable in the mining industry, almost readily achievable, if not achieved; I've very little doubt about that.

But I think that, to apply those standards to a textile industry, would be misleading. I think equally misleading would be to expect that the mining industry should work to the standards of the textile industry. That's the problem. That's why I emphasize this issue.

I know that legislation likes to have a nice, simple thing; a standard for asbestos should be two, half, one, whatever. That might be convenient, but it is ignoring a lot of information.

Q. I guess one of our concerns is, when one speaks of the overall excess mortality, if we compare the SMR's that you get these percentages from, on the top paragraph, righthand side, of page 12, where the SMR is 1.02 and 1.10 respectively -- if one compares that to the general population, one would come up with an overall excess mortality of two or ten per cent, as you've done.

But if one compares that with the SMR's that one would expect in working populations (as you've mentioned, the healthy worker effect), which usually run from about point six to point eight, or somewhere in that ballpark, this excess mortality would then be significantly higher; not two or ten per

Q. (cont'd.) cent, but twenty or thirty per cent. Would you comment on that.

A. I don't think that is true. I think studies of healthy worker effect have shown that that sort of difference is only for perhaps the first ten years of employment, but that, by the end of around ten years of employment, the healthy worker effect almost goes.

Indeed, bearing in mind that our more detailed analyses here are based on workers at least twenty years from first employment, it is — the contribution of the healthy worker effect is probably not very significant. It's probably there and it isn't just that it should be thought about; it is that one should look very cautiously at any data which compare a work populations group with the population of Quebec. Any group of workers is different from that, and it's only just a useful guide; that's all.

Q. Are you saying that, after ten years of work, for most working populations, the SMR approach is one?

A. Yes; yes. It approaches it -- in cancer, there's very little -- as a matter of fact, there's relatively little evidence of the health worker effect in relation to cancer at all, which, when you come to think of it, is not surprising, because, after all, what causes cancer is not something which is going to influence whether you're employed or not.

On the other hand, if you have a -- what shall we say? -- chronic heart disease, or a chronic respiratory disease, your chance of getting a job in the mines is less; and, therefore, in the next, shall we say, five or ten years after employment, or, shall we say, from that date onwards, if you took some workers and you accepted some and rejected others, the ones you rejected would have a much higher mortality for ten years after that assessment than the ones who were accepted.

A. (cont'd.) But in cancer this is not too evident.

Q. Okay.

I was interested in what, if any, conclusions one could draw from this paper with regard to other aspects of asbestos exposure for working people, in that it seems that you said today, in addition to some other people, I'm told, who were here earlier, though I wasn't, that less asbestos is seen in mining, as a rule, in asbestos miners, than in populations involved in production as one sort of goes upstream.

And I guess my question is, how applicable do you think -- how applicable would conclusions be drawn from this study, based on a dose-response, would those be to other parts of the industry, in terms of protecting workers?

A. I think that -- I think it is useful information to take into account, but I think that if you ignored other information, it obviously would be inadequate.

In 1964, at the New York meeting on asbestos, the first sort of international meeting on this subject, it was universally agreed that everybody would go away and study all the various industries in this way. If everybody had, we would be able to answer that question better.

It's unfortunate that we who did should thereby be criticized for not having answered all the other questions. We didn't; we looked at the mining industry. And I'm sorry if it doesn't answer all the other questions.

But, of course, there are beginning to be some other studies now, at long last, of what the health experience in some of the other industries is.

Q. So, are you saying that, if one generates a dose-response curve from a study that's based on a mining population, such as you've done, that you don't feel it's fair to

Q. (cont'd.) extrapolate that to other parts of the industry?

A. Only some of it. I find it is useful; I think it likely that if you have a linear relationship in the mining industry, you will probably have a linear relationship in other industries. Now, that's a very substantial piece of information -- I mean, it didn't exist before that.

I think that the interactions with smoking are also very important to note. I think the absence of mesotheliomas in the chrysotile industry are important to note. I think the fact that asbestosis, even in mining, where we can say it looks as if the lung cancer risk is appreciably less than, say, in the asbestos textile industry, it is interesting, nevertheless, that asbestosis is quite common in the mining industry.

So, in other words, I think you have certain pieces of information; you can't simply -- in fact, strongly recommend that nobody does try and take this relationship and say it applies to another one. But it seems to me that the principles involved may, to a degree, apply.

And, therefore, it means that if you have an inadequate study somewhere else (and there are a number of those), you can perhaps -- it may help you to say, well, we haven't got a proper study in the asbestos cement industry, but still we've got some study.

Now, probably the principles of the mining industry still apply there, and we may be able to make more of that inadequate study than we could have done otherwise; it's that kind of thing.

I mean, I'm sure with all this we've got to use all the data there is, and then make up our mind which is sensible.

Q. Well, it's getting near 5:30, but maybe if I

Q. (cont'd.) could ask one more question; reading your paper, it appears to me that the expected lung cancer rates that you use in comparing the rates in your cohort was with the Province of Quebec; is that correct?

A. Yes, it is. Well, we used three methods of analysis, and that was one of them; yes.

Q. Now, I'm wondering; shouldn't one compare such a rural problem -- wouldn't the ideal comparison be with another rural population non-exposed to asbestos as opposed to a population in Quebec where I'm told the lung cancer rates do exceed rural rates by as much as thirty per cent?

A. I think that there is something in that. The problem is to get comparative rates from anywhere else; they don't exist, unfortunately.

I certainly think that it is better, shall we say, to use the Province of Quebec than the whole of Canada, or, as has been done in American studies, using the whole of the United States' population for comparing.

I think that obviously, within reason, the more similar, the better. But this was one of the reasons which, indeed, we have written at some length on how critical feel of the use of such reference populations, and why we think that, really, the use of the -- what we call the internal case reference type of analysis is probably fairer. It probably gives you a better indication of the pattern and relative risk.

It admittedly, I think, as the Chairman already mentioned -- it does presuppose; it is based upon the concept that the very low levels carry, essentially, a zero risk, and that is an assumption, but it's an assumption that one can look at critically and say, "What sort of a correction would you like to make for that?" But it's very, very difficult.

A feature of a number of epidemiological studies,

A. (cont'd.) which perhaps should be mentioned, is that low-exposure people have funny features. There are a number of cohort studies which show that people who are in an -- see; one of the groups of people that have a low exposure are people who are in, for a short time and for whatever reason -- in low-exposure jobs, and one of the reasons is, of course, that they're not necessarily very fit. And one of the reasons they're not fit is because they smoke too much. I mean, that's a very common thing.

So it's not uncommon to find that, in any kind of array of figures, you find to your distress the lowest-exposure group has got a relatively higher lung cancer rate. We found that in this study of a gold mine, too; and I think other people have found this.

So this could be another factor which is sort of undesirable. It's really -- you know, you can't really expect too much from epidemiology --- what you can get out of it is the shape of the distribution and some sort of concept of what the relative risk is at higher levels compared with low ones. But I don't think it should be taken beyond that.

Q. Well, you know, our concern is that, in comparing a rural population with the more urban population, that the expected rates might be inflated, and therefore would mask a real increase among the rural population being studied; in this case, the miners.

As you know, the NCI in the United States has put together a whole series of cancer maps which show quite clearly that the rates of cancer are increased in the urban areas as opposed to the rural areas.

A. Yes. What they don't show is why.

You see, one important aspect of this is that cancer is one of those diseases that is better diagnosed in

A. (cont'd.) hospital, where there are hospital facilities. And I think there's very little doubt that cancer is less completely recognized in rural areas.

So we thought about this quite a lot in relation to the Asbestos and Thetford working populations, because you've got to bear in mind that they didn't necessarily die in Asbestos and Thetford. In fact, a very high proportion did not die in Asbestos and Thetford; they died in Montreal, Quebec City, Sherbrooke. Because, you see, most people die after they retire; not very much of the mortality is actually in the work age, it's later, by which time there's quite a lot of migration to the urban areas.

The other point is that, in Asbestos and Thetford, although they are small-town cities in a rural area, they do have moderately developed medical facilities. So I don't know; it could be that they're over-diagnosed or under-diagnosed; I don't know. That's the problem: I don't know the correct basis.

Q. But I think we can agree that there is a significant difference in -- you know, in cancer incidence, if one compares rates in a more urban population, such as the Province of Quebec, with a rural area; that's the point that I'm trying to get clarified.

A. This is true; but it might not apply to the asbestos workers. Because, you see, after all, one of the things they are subject to is a much higher level of medical supervision than the population of that district; much higher level.

DR. NELSON: Okay.

DR. DUPRE: You will have the opportunity to complete your questioning beginning at 9:00 tomorrow morning.

DR. NELSON: Thank you.

DR. DUPRE: Dr. McDonald, thank you so very much. Class is dismissed until 9:00 a.m. tomorrow.

INQUIRY ADJOURNED

THE FOREGOING WAS PREPARED
FROM THE TAPED RECORDINGS
OF THE INQUIRY PROCEEDINGS

DEREK WEST